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August 15, 2018

*Via electronic submission to https://oehha.ca.gov/comments*
Monet Vela
Office of Environmental Health Hazard Assessment
P.O. Box 4010
Sacramento, California 95812-4010

Re: Proposed Adoption of New Section Under Article 7: No Significant Risk Levels
Section 25704: Exposures to Listed Chemicals in Coffee Posing No Significant Risk

**CERT'S SUBMISSION NO. 1**

Dear Ms. Vela:

Enclosed herewith are the following documents that are being submitted on behalf of our client, the Council for Education and Research on Toxics (CERT) regarding testimony that the coffee industry's nutritional epidemiology expert, Dr. Dominik Alexander, gave during the Phase 2 trial in the *CERT v. Starbucks* case explaining why he could not say that the inverse associations reported between consumption of coffee and various cancers and chronic diseases are causal and that no health benefit could be ascribed to coffee consumption in the absence of a causal association.

1. Exhibit A - Testimony of Dr. Dominink on cross-examination in *CERT v. Strarbucks* trial, September 7, 2017 a.m.

2. Exhibit B - Curriculum Vitae of Dr. Dominik Alexander.

Kindly include these materials regarding Dr. Dominik Alexander in the record for this rulemaking proceeding.

Very truly yours,

Raphael Metzger

RM:ip
encls: as specified

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# EXHIBIT “A”

SUPERIOR COURT OF THE STATE OF CALIFORNIA

FOR THE COUNTY OF LOS ANGELES

DEPARTMENT 323 HON. ELIHU M. BERLE, JUDGE

| | |
|---|---|
| CERT,<br><br>Plaintiff,<br><br>vs.<br><br>STARBUCKS CORP, ET AL.,<br><br>Defendants. | SUPERIOR COURT<br>CASE NO. BC 435759<br>BC 461182 |

REPORTER'S TRANSCRIPT OF PROCEEDINGS

Thursday, September 7, 2017

(A.M. Session)

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(Appearances continued on next page.)

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(A.M. SESSION)

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M - Mr. Metzger

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CASE NUMBER: BC 411192/BC435759
CASE NAME: CERT CASES
LOS ANGELES, CALIFORNIA THURSDAY, SEPT 7, 2017
DEPARTMENT 323 ELIHU M. BERLE, JUDGE
REPORTER: DAVID A. SALYER, CSR 4410
TIME: 9:00 A.M.

-o0o-

THE COURT: Good morning, counsel.

Back on the record in the case of CERT versus Starbucks.

All counsel are present and Dr. Alexander is on the stand.

Is Dr. Alexander here?

MR. KENNEDY: Yes, he is, your Honor.

Your Honor, before we get started, it turns out my attempt to save some court time yesterday was well intentioned but badly executed.

I would request leave to re-open direct examination for the limited purpose of having Dr. Alexander formally read into the record the various diseases and conditions listed on Exhibit 73528 and 73529 for identification.

THE COURT: Do we have do that?

Can't we reach a stipulation with regard to that information and just have that document marked in evidence, not for the truth of the matter, but that his testimony will be the identification of those diseases?

Is that satisfactory?

MR. METZGER: I have a concern. Perhaps weak, but

here's the concern.

THE COURT: Yes.

MR. METZGER: Once expert's opinions are -- written opinions are admitted in evidence, I think it needs to be across the board. They're all hearsay.

So I don't want to go onto a slippery slope.

THE COURT: Everything is hearsay.

MR. METZGER: I know. I don't want his opinions to be marked as exhibits and admitted into evidence where plaintiffs are not.

THE COURT: No, it's not intended to be his opinion.

Otherwise, Mr. Kennedy is just going to ask him are these all these diseases and he'll recite it, and we'll lose ten minutes. Although we're losing ten minutes just talking about it.

If you want him to just read a list and then the next witness will read his or her list.

MR. METZGER: I understand.

If it's merely going to be a list of -- so what exactly is it that you want to have admitted?

MR. KENNEDY: It's Exhibits 73528 and 73529, which are slides 21 and 22 that are labeled respectively "No independent association."

THE COURT: All right. Why don't we order counsel to meet and confer to see if you can agree to it during a recess.

In the meantime, let's have Dr. Alexander resume the stand.

I'd suggest that a number of exhibits -- there may be

other areas too that counsel could meet and confer and shorthand the testimony so you can reach a stipulation and certain exhibits can be admitted just for informational purposes, and in fact they are demonstrative evidence of what the witness has said or will say or just something that's not disputed with regard to what he would say, not for the truth of anything set forth. Because there's a hearsay problem with all the testimony.

Not a problem, but experts testify from hearsay information. These articles are all hearsay and the witnesses' opinions and expressions of their analyses done at other times is all hearsay, anyway.

So I will ask counsel to meet and confer about that, see if you can resolve it.

Dr. Alexander, do you understand you're still under oath?

THE WITNESS: Yes, your Honor.

DOMINIK DANE ALEXANDER,

witness, resumed the stand and testified further as follows:

THE COURT: Please resume the stand and restate your name for the record.

THE WITNESS: Dominik Dane Alexander.

THE COURT: Mr. Metzger is inquiring.

Just one second. I want to make sure I have the LiveNote up and running here. One second.

///

///

CROSS-EXAMINATION (Continued)

BY MR. METZGER:

Q. Good morning, Dr. Alexander.

A. Good morning. How you doing?

THE COURT: Just one moment. I'm trying to get the LiveNote working.

MR. METZGER: I apologize.

(Pause in proceedings.)

THE COURT: All right. We're live.

Mr. Metzger, you may proceed.

MR. METZGER: Thank you, your Honor.

Q. Dr. Alexander, since Mr. Kennedy just raised this issue, I would like you to take a look at what is identified as Exhibit 73528 within your binder.

It's slide 21.

A. Yes, sir.

Q. All right. And there's a title for this slide, which is "No independent association," correct?

A. That is correct.

Q. All right. Have you ever seen the term "independent association" defined in any textbook of epidemiology?

A. I believe I have at some point.

Q. Can you identify any textbook of epidemiology that defines that term that you have used?

A. I don't recall specific textbooks. It's a common term used in epidemiologic practice.

Q. Okay. And can you cite me any published

peer-reviewed article or any textbook that actually defines that term that you have used, "independent association"?

A. Again, I don't recall that any actually define it.

I know that they do, but, again, in epidemiologic practice that's a commonly used term.

Q. Okay. So yesterday we were talking about some of the work that you have done that's been sponsored by various companies.

You have actually also, on behalf of food companies, advocated that certain chemicals not be listed by the State of California as carcinogens, true?

A. What do you mean by advocated?

Q. Where you've submitted material to the agency saying you don't think that a particular chemical in food should be listed as a carcinogen.

You've done that, haven't you?

A. I've reviewed the evidence. I don't recall a specific situation.

Q. All right. I've provide you what we're marking as exhibit --

MR. METZGER: Who is the defense counsel who gets these now?

MR. MARGULIES: Mr. Kennedy.

MR. METZGER: Okay.

Q. I'll provide you what we are marking as Exhibit 61837.

(Exhibit 61837, Document, marked for I.D.)

Q. BY MR. METZGER: It is a document that's dated October 17, 2016. It's titled "Comments of California League of Food Processors, California Retailers Association, California Chamber of Commerce, California Grocers Association, Western Agricultural Processors Association, Grocery Manufacturers Association and North American Meat Institute regarding whether nitrite in combination with amines or amides has been clearly shown through scientifically valid testing according to generally accepted principles to cause cancer."

And this is signed by you, is it not?

A. I did review the epidemiology on nitrite and cancer. I did write a section.

Q. My question is, is that your signature on the last page of this document?

A. The very last page, yes, it is.

Q. Right. And right above that signature it's also signed by J. Murray, who you know, correct?

A. By phone only.

Q. Okay. All right. And right above both of your signatures, it says, "For all the above reasons, nitrite in combination with amines or amides has not been clearly shown to cause cancer."

That's what you were telling the State of California on behalf of all these food organizations, not to list it, correct?

A. That is a review of the epidemiology and based on the epidemiology, there is no independent association.

Q. So the answer to my question is true, is yes, correct?

A. True, yes.

Q. All right. Fine.

Now, you have also testified on behalf of companies in litigation, have you not?

A. I have.

Q. All right. And you began testifying for companies in litigation in July of 2014, right?

A. I believe so, yes.

Q. Right. And that was after you participated in an asbestos medicine seminar sponsored by the Defense Research Institute in November of 2013, correct?

A. In terms of the timeframe, but not a cause-and-effect relationship.

Q. Okay. I know you're not testifying about causation. I got that. Okay.

And the Defense Research Institute is the leading organization of defense attorneys and in-house counsel in the United States, correct?

A. I am not sure. That may be how they describe themselves.

Q. Okay. You've seen their website where they describe themselves as the voice of the defense bar?

A. I think you've raised that before.

Q. Other attorneys have raised that with you?

A. So in light of that.

Q. Correct?

A. Yes.

Q. And at that November, 2013 Defense Research Institute asbestos seminar, asbestos medicine seminar, you met a Mr. Bouchard, who is an asbestos defense attorney, correct?

A. Yes.

Q. And he hired you to testify on behalf of asbestos defendants in asbestos litigation, correct?

A. I have worked with Mr. Bouchard on a few occasions.

Q. He's hired you, hasn't he?

A. I've been retained on behalf of his clients, yes, in asbestos litigation matters.

Q. Is there a difference between being retained and being hired?

THE COURT: Let's not quibble. Let's move on.

THE WITNESS: I'm not sure.

Q. BY MR. METZGER: Okay. Let's not quibble. All right.

So after Mr. Bouchard hired you, you began testifying in asbestos cases at deposition and I think also at some trials, correct?

A. I have testified in a couple of asbestos trials, not with Mr. Bouchard.

Q. So you've now testified -- you now give about 20 depositions a year. You testify at about 20 depositions or trials a year?

A. Perhaps. It sounds reasonable.

Q. And most of those are asbestos cases, correct?

A. Yes.

Q. Okay. Let's -- I'll provide you what's been marked as Exhibit 60224.

This is a list of your testimony, is it not?

A. It is.

Q. Okay. And this is a complete list of your testimony, is it not?

A. As of June 5th.

Q. Okay. All right. And in every one of the cases on this list you've testified on behalf of the defendants, correct?

A. That is correct.

Q. Okay. And every one of these cases that you've testified, you were retained by lawyers representing defendants in litigation, correct?

A. That is correct.

Q. Okay. And in the asbestos litigation you rendered two opinions;

One, that the available epidemiologic evidence does not support an increased risk of mesothelioma among motor vehicle mechanics and those involved in brake repair, correct?

A. Yes.

Q. And the other is that the available epidemiologic evidence does not support an increased risk of mesothelioma among individuals exposed to low or moderate levels of chrysotile asbestos, correct?

A. Yes, I've testified to that.

Q. Okay. All right.

One other thing.

Now, as an epidemiologist, have you actually conducted some epidemiologic studies?

A. I have.

Q. Okay. And have you conducted or performed any case control studies that evaluated coffee as a factor?

A. No, I have not.

Q. And have you published any cohort studies that have evaluated coffee as a factor?

A. No.

Q. Any randomized controlled trials?

A. No.

Q. Are you able to identify any publication that you have written that actually mentions coffee?

A. I don't recall.

I may have. I'm not sure.

Q. And are you able to identify any publication that you have written that actually mentions acrylamide?

A. I don't believe so.

Q. Okay. Yesterday we spoke briefly about the International Agency for Research on Cancer and their update evaluation for coffee.

Do you recall that?

A. I do.

Q. And do you recognize the International Agency for Research as the authoritative or reputable scientific organization for the evaluation -- for the identification of carcinogens?

A. I do recognize IARC as a reputable source.

Q. Okay. Have you ever personally been a member of an IARC Working Group for any evaluation of any of the substances that they have evaluated?

A. Not as a Working Group member.

Q. Okay. But you have attended some of those meetings as a representative on behalf of industry, correct?

A. I have.

Q. Correct.

All right. So I have a proposition for you. I asked it to you in your deposition and I'll ask it to you now.

A. And I said it was 65?

THE COURT: All right. Let's stop the chitchat. Ask a question.

Q. BY MR. METZGER: My question is, is it true that in every instance where you have evaluated the carcinogenicity of a chemical or an agent, you have concluded less carcinogenicity than IARC?

A. I don't think that's necessarily accurate.

Q. Okay. So let's go to the first slide.

One of the substances you had evaluated is trichloroethylene, correct?

A. Yes.

Q. And that's a chlorinated solvent, right?

A. It is.

Q. And you are an author of an article, A Review of Trichloroethylene and non-Hodgkins lymphoma from 2006, right?

A. That is correct.

Q. Which was, what, 11 years after IARC issued its monograph on trichloroethylene in 1995, right?

A. Yes.

Q. At that time IARC concluded that, "Trichloroethylene is probably carcinogenic to humans. Several epidemiologic studies showed elevated risks for non-Hodgkin lymphoma."

That's IARC 1995. And in 2006 you wrote, "Although a modest positive association was found in the TCE subcohort analysis, there is insufficient evidence to suggest a causal link between TCE exposure and NHL."

That was your assessment, right?

A. Correct, yes.

Q. All right. Next slide.

Trichloroethylene and liver cancer. You wrote the article on liver cancer in 2007, at which time -- well, let's go back to IARC 1995 regarding liver cancer.

IARC wrote that several epidemiologic studies showed elevated risks for cancer of the liver and biliary tract.

It was probably carcinogenic to humans.

In 2007, 12 years later, you wrote, "The current epidemiologic data are not sufficient to support a causal relation between occupational TCE exposure and liver/biliary cancer," correct?

A. It is. We're talking about risks and causation.

My opinions were actually in concert with IARC at that time.

Q. Next slide.

Trichloroethylene and kidney cancer.

The National Toxicology Program in the 11th Report on Carcinogens in 2004 concluded that:

> "Trichloroethylene is reasonably anticipated to be a human carcinogen and that a meta-analysis of seven cohort studies found that occupational exposure to TCE was associated with excess incidences of liver cancer, kidney cancer, non-Hodgkins lymphoma, prostate cancer and multiple myeloma, with the strongest evidence for the first three cancers."

And then IARC in 2014 said:

> "There is sufficient evidence in humans for the carcinogenicity of trichloroethylene. Trichloroethylene causes cancer of the kidney."

MR. KENNEDY: Your Honor, object under 720. It has not been established that these are materials he read, considered or relied upon or that they've been independently established as authoritative and introduced into evidence.

THE COURT: Overruled.

Q. BY MR. METZGER: Actually, you've reviewed all these, haven't you?

A. I'm familiar with them. I think some of these quotes are taken out of context.

Q. Okay.

A. I actually agree that there are increased risks.

Q. Okay. And in 2010, regarding trichlorethylene and kidney cancer, you concluded:

"Positive associations were observed across various study groups. However, considerations of unmeasured potential confounding, lack of quantitative exposure assessment and lack of exposure response patterns limit epidemiologic insight into the role of trichlorethylene exposure and its potential causal association with kidney cancer."

Right?

A. Yes. Four years prior to IARC, yes.

Q. By the way, do you now agree that trichlorethylene causes kidney cancer?

A. I believe that there are positive associations. Just like IARC and the ROC report, there are positive associations for liver cancer and NHL. So I am in agreement with IARC.

However, there is a recent large-scale study just published in Sweden that actually shows an inverse association with TCE and kidney cancer.

Q. But I don't think you answered my question.

My question is, do you now agree with IARC that trichlorethylene causes cancer of the kidney?

I'm not talking association. I'm talking causation.

Do you agree with IARC now?

A. I would have to go back and revisit all the

current evidence. My review was in 2010, so I would need to evaluate it now.

Q. All right. That's fine. Arsenic in drinking water and bladder cancer.

IARC, 2004. IARC says, "There is sufficient evidence in humans that arsenic in drinking water causes cancers of the urinary bladder."

You four years later, "Although uncertainties remain, low-level arsenic exposure alone did not appear to be a significant independent risk factor for bladder cancer."

That was your conclusion after IARC had concluded causation, correct?

A. You're taking this out of context.

IARC is referring to specific subpopulations of endemic areas, largely of Taiwanese study populations who were malnourished.

So I do think at very high levels, yes, arsenic in drinking water can cause bladder cancer, but that's not what I'm referring to in my evaluation.

Q. Okay. Next slide.

Processed meat and colorectal cancer.

IARC in 2015, "A meta-analysis of colorectal cancer in ten cohort studies" -- which is Chen, 2011 -- "reported a statistically significant dose-response relationship with a 17 percent increased risk per 100 grams per day of red meat and an 18 increase per 50 grams per day of processed meat."

Five years earlier you conclude, "The current available epidemiologic evidence is not sufficient to support a clear

and unequivocal independent positive association between processed meat consumption and colorectal cancer."

That was your conclusion, correct?

A. It is. And I had generally the same findings as Chen did, so we're definitely in concert.

Q. Next slide.

So red and processed meat and prostate cancer.

IARC, 2015, "Positive associations were seen in cohort studies and population-based case control studies between consumption of red meat and cancers of the prostate."

You conclude, quote, "The results of this meta-analysis are not supportive of an independent positive association between red or processed meat intake and prostate cancer."

That was your conclusion, correct?

A. Yes. But you've mixing apples and oranges here.

MR. KENNEDY: Your Honor, the witness is entitled answer questions. He's gotten interrupted on the last four.

THE COURT: Counsel, give the witness an opportunity to answer, and the witness will give counsel the opportunity to finish the question.

MR. METZGER: Go ahead.

THE COURT: Did you complete your last answer?

THE WITNESS: I think, your Honor, what I was saying is it's a different comparison.

We actually concluded the same thing. There are positive associations, but just like this matter here, there is not an independent relationship.

Once again, we are in concert, and IARC in 2015

actually was referring to some of my research when they made that statement.

MR. METZGER: Next slide.

Q. All right. So benzene and non-Hodgkin lymphoma. IARC in 2012, "There is sufficient evidence in humans for the carcinogenicity of benzene, although also a positive association has been observed between exposure to benzene and non-Hodgkin lymphoma."

Your conclusion, 2010, "The results of this meta-analysis are not supportive of an independent association between benzene exposure and non-Hodgkin lymphoma," correct?

A. That's what's indicated. And, again, our conclusions are consistent regarding associations.

Q. And the meta-analysis that's referred to there is your meta-analysis, correct?

A. On the right of the screen?

Q. Yeah.

A. The Alexander 2010?

Q. Right.

A. Yes.

Q. All right. Next slide.

Ingested nitrate and nitrite in stomach cancer.

So this is the subject you wrote to the State of California with J. Murray, correct?

A. Yes. The one you provided, yes.

Q. So IARC in 2010 concludes, "Ingested nitrate or nitrite under conditions that result in endogenous nitrosation is probably carcinogenic to humans. Nitrite in food is

associated with an increased incidence of stomach cancer."

Two years later you write, "Newly published prospective epidemiological cohort studies indicate that there is no association between estimated intake of nitrite and nitrate in the diet and stomach cancer."

That's what you conclude, right?

A. Yes. I updated the state of the epidemiologic science in IARC's assessment, and clearly there is no association.

That was discussed in Lyons, France, when I was there at the IARC meetings as well.

Q. Next slide.

Low dose arsenic exposure and bladder cancer. IARC, 2012.

> "Arsenic and inorganic arsenic compounds are carcinogenic to humans. The observed association between exposure to arsenic in drinking water and bladder cancer cannot be attributed to chance or bias. There is evidence of dose-response relationships within exposed populations."

Your review states:

> "The consistent results for never smokers, in particular, indicate that low-level exposure to arsenic in drinking water alone is unlikely to contribute to an increase in bladder cancer incidence."

That was your conclusion, correct?

A. Yes. That's what I wrote.

Q. Right. Next slide.

That's it. Okay. Excuse me one second, your Honor.

All right. So you were hired for this case after you were contacted by the defense, by Michele Corash, correct?

A. I believe so.

Q. And your retention letter is dated March 23, 2017, correct?

A. I understand that to be correct.

Q. Right. So you began your work about then and have continued working on this case ever since, correct?

A. Yes.

Q. And your deposition in this case took place on -- let's see. That was -- do you recall the date?

A. June.

Q. June 7. Okay.

So within about ten weeks, after being retained, you reviewed materials and gave your deposition, correct?

A. That sounds accurate.

Q. Okay. Now, before -- and actually, again, you got working on this project in April; is that right?

A. It would have been sometime after the engagement letter, I believe.

Q. All right. Before April of this year, had you systematically reviewed the epidemiologic studies regarding coffee and cancer?

A. Not systematically.

I have reviewed many of them.

Q. Prior to April of this year, had you systematically reviewed the epidemiologic studies regarding coffee and chronic diseases?

A. Same response. I've read them but not systematically.

Q. Okay. Prior to April of this year, had you systematically reviewed the epidemiologic studies regarding acrylamide and cancer?

A. No.

Q. Have you done that to this date?

A. No.

Q. Okay. So between -- I'll provide you with Exhibit 60226.

This exhibit is an invoice dated May 18th for the work that EpidStat, your employer, did for this case, correct?

A. Yes, as of this date.

Q. But actually this is just an invoice for your services, correct?

A. Incorrect.

Q. Okay. I see. The second page has others.

So there were other people working on this with you at EpidStat?

A. That is correct.

Q. How many others?

A. Three or four or five.

Q. So for the first invoice, which was through the end of April of 2017, EpidStat billed the defense in this case 34,700 odd dollars, correct?

A. That's what's indicated, yes.
Q. All right. And how much additional work did you do on this case between that first invoice and the date of your deposition?
A. How much additional work in terms of hours --
Q. Hours.
A. -- for myself?
Q. Yeah.
A. I don't recall the specific hours.
A few dozen, I would say, at least.
Q. At your deposition you said 60 to 80. Does that sound about right?
A. That could be, yes.
Q. Okay. And how many hours for the other workers at EpidStat?
A. I would estimate the same.
Q. Okay. And since your deposition until today, how many hours have you spent on the case?
A. Since my deposition until today, probably closer to that 60-hour mark, again.
Q. Okay. All right. And how much are you charging for your services?
A. 390.
Q. All right. Now, do I recall correctly that at some point in your career you assisted one of the defendants in this case in obtaining approval or authorization of a qualified health claim?
A. Can you repeat that?

Q. Do I recall correctly that at some point in your career you helped one of the defendants in this case, Nestle, obtain an authorization from the FDA for a qualified health claim?

A. I have worked with Nestle on a qualified health claim in the past.

Q. Okay. And when you say you worked with them, you presented information to the FDA to help Nestle obtain authorization for a qualified health claim, correct?

A. In general, yes.

My role was to review the epidemiology and I assisted Nestle in that process.

Q. Right. And in that context you became familiar with the FDA's guidance for industry, the evidence-based review system for the scientific evaluation of health claims, correct?

A. I was already familiar with the process.

Q. Oh, okay. Good.

I will provide you Exhibit 59070.

And this exhibit is -- you recognize this, do you not?

A. Yes.

Q. And you studied this and became familiar with it, at least in the context of that work that you did for Nestle, correct?

A. I'm familiar with this, yes. I've reviewed it.

Q. Okay. I would like you to turn to the fourth page of this document.

There is a section 3 entitled Evidence-Based Review

System for the Scientific Evaluation of Health Claims?

A. I am there.

Q. And under this section there's a heading, What Is an Evidence-Based Review System.

Do you see that?

A. I do.

Q. And it says:

"An evidence-based review system is a systematic science-based evaluation of the strength of the evidence to support a statement. In the case of health claims, it evaluates the strength of the scientific evidence to support a proposed claim about a substance/disease relationship."

Do you see that?

A. I do.

Q. And you agree with that, don't you?

A. I would say that for a health claim, for the purpose of selling a product and putting a label on a product, and in the context of what the FDA's guidance is, they are discussing evaluating the strength of the evidence to support putting a label on a product that's being sold.

Q. Okay. And they're also discussing systematic reviews, correct?

A. In the context of their process for a health claim, yes.

Q. And you do systematic reviews, do you not?

A. I do.

Q. And the systematic reviews that you do are evidence-based, true?

A. I would like to think scientifically everything I do is evidence-based.

Q. Okay. Now, the last sentence in the paragraph says, quote:

> "After assessing the totality of the scientific evidence, FDA determines whether there is SSA to support an authorized health claim or credible evidence to support a qualified health claim."

Do you see that?

A. I do.

Q. And SSA is referring to -- it's an acronym for significant scientific agreement, correct?

A. Yes. That's my understanding.

Q. Do you agree that in determining whether there is significant scientific agreement to support a health claim, that endeavor should be done after assessing the totality of the scientific evidence?

MR. KENNEDY: Inadequate hypothetical. It's not clear whether it's being restricted to somebody trying to make a claim on a product or somebody else.

THE COURT: Overruled.

THE WITNESS: I think that certainly depends on the scientific exercise we're talking about here.

Q. BY MR. METZGER: Okay. Would you turn to the next page.

In the middle of the page there is a paragraph that begins with the language "for example."

Do you see that?

A. I do.

Q. And it says, "For example, cancer is a constellation of more than 100 diseases," and it goes on.

Do you agree with that?

A. In general, I think, based on subtypes of cancer, yes. There are more than 100 different types of unique cancer subtypes.

Q. The next sentence says, quote, "Cancer is categorized into different types of diseases based on the organ and the tissue sites." Is that true?

A. Yes. Certain organizations categorize cancer by organ and tissue sites, yes.

Q. And then it says, "Cancers at different organ sites have different risk factors, treatment modalities and mortality risk."

Do you agree?

A. Many do, yes.

Q. And then in the middle of that paragraph, about the seventh line down, there's a sentence that says, "The etiology, risk factors, diagnosis and treatment of each type of cancer are unique."

Do you see that?

A. I see what you're reading from.

Q. Do you agree with that?

A. That certainly depends.

Q. All right. The next sentence -- the latter part of the sentence says, quote:

"FDA's current approach is to evaluate each form of cancer individually in a health claim or qualified health claim petition to determine whether the scientific evidence supports the potential substance/disease relationship for that type of cancer."

Do you see that?

A. I see where you're reading from.

Q. In doing your scientific evaluations of substance/disease relationships, do you evaluate each form of cancer individually?

A. I do.

I'm sorry. You're referring to this particular matter or in general? I have also looked at total cancer for certain research projects, and I also look at specific cancers.

Q. Okay. All right. Just a second.

Now, if you look at the very last sentence on page 5 of this document, it says, "Randomized controlled trials offer the best assessment of a causal relationship between a substance and a disease because they control for known confounders of results."

Do you agree with that?

A. It certainly depends on the scientific application.

Q. The theoretical basis is there, isn't it?

A. The theoretical basis is there, but, again, it

certainly depends on how it's being applied.

That's the most important part of it.

Q. You understand that language is used almost universally when it comes to scientific evidence, true?

A. I --

MR. KENNEDY: Objection, vague and indefinite.

THE WITNESS: That language is used by some and in different situations.

But, again, it certainly depends.

MR. METZGER: I'll read from the witness's deposition at page 39, lines 13 through 22.

Any objection?

MR. KENNEDY: 39?

THE COURT: Do I have a copy of the deposition up here?

MR. KENNEDY: Your Honor, can I inquire, again, which lines you're talking about?

THE COURT: Just one second. Page 39, lines 13 to 22.

MR. KENNEDY: Your Honor, I do object.

I see, for starters, it stops in the middle of the answer.

THE COURT: 39. Let's see.

Yes. Let's start with the previous question.

I think you have to go back to 38, line 20, to make an understanding of this --

So it's 39, line 1 through --

MR. KENNEDY: Your Honor, I would ask to go through line 40, line 4, to complete the sequence.

THE COURT: I'm sorry?

MR. KENNEDY: I'd request that the reading go through line 40, line 4, to complete the sequence.

THE COURT: It goes on and on.

Let's read to 40 -- the beginning of 38, line 20 to 40, line 4.

MR. KENNEDY: With that, your Honor, I would object. It's not impeaching.

THE COURT: Mr. Metzger, you may read it.

MR. METZGER: You want me to read from 38, line 20?

THE COURT: Yes.

MR. METZGER: All right.

"Q. Have you ever published any study regarding total cancer?

"A. I believe I have evaluated total cancer, at least total cancer mortality, in a prior review or meta-analysis.

"Q. What is that?

"A. If I recall, I believe that was on dietary supplements, multivitamin supplement use. So that would have been total cancer in addition to other mortality.

"Q. And did that also evaluate individual cancers?

"A. I don't recall. I do recall cardiovascular disease, total mortality and total cancer.

"Q. Got it.

Any other publications that you have done regarding total cancer?

"A. There may have been where I have reported risk estimates for total cancer. I don't recall right now.

"Q. Okay. Look at the last sentence on the page of this document, which is Exhibit 2. It says, 'Randomized controlled trials offer the best assessment of a causal relationship between a substance and a disease because they control for known confounders of results.'

Do you agree?

"A. Yes and no. I think that is a pretty broad characterization of randomized controlled trials.

I understand that language is used almost universally when it comes to scientific evidence."

You wanted me to read further to where?

Okay. (Reading:)

"A. But there is some specific nuances to RCTs and regarding causal relationship and their control of confounding that I would be happy to discuss.

"Q. So you generally agree with that statement?

"A. The theoretical basis is there. However, the pragmatic aspects for specific topic areas may not be relevant when it comes to RCTs."

Q. All right. Now, the last sentence of this paragraph says, "Therefore randomized controlled intervention studies provide the strongest evidence of whether or not there is a relationship between a substance and a disease."

Do you agree?

A. I think it certainly depends on, again, the scientific application to that.

Q. Okay. Where randomized controlled intervention studies have been done, do they provide the strongest evidence of whether or not there is a relationship between a substance and a disease?

A. Again, it certainly depends on how they were applied and what topic area that we're talking about.

Q. Are you aware of any instance where an epidemiologic study type was found to provide stronger evidence for a substance/disease relationship than the randomized controlled intervention study where that had been done?

A. What do you mean by stronger evidence? What situation?

Q. You use the term "stronger evidence" all the time. So use your own definition in answering the question.

A. Well, are you referring to strengths of association in this context or the sufficient quality of

evidence?

Q. You were able to answer this question at your deposition, weren't you?

A. I believe so.

Can you repeat it one more time for clarification?

Q. Should I just read the answer at your deposition? Would that be better?

A. However you want to do it.

Q. Let's do that. Okay.

"A. --

MR. KENNEDY: Can we have page and line number, please?

THE COURT: I'm sorry.

MR. KENNEDY: Your Honor, could we have a page and line number, please?

THE COURT: Yes.

MR. METZGER: I'll read from page 42, line 23, through page 43, line 11.

THE COURT: Any objection?

MR. KENNEDY: I don't believe it's impeaching, but no objection to it being read.

THE COURT: All right. Mr. Metzger, go ahead.

MR. METZGER: (Reading:)

"Q. Well, are you aware of any instance where an epidemiologic study type was found to provide stronger evidence for a substance/disease relationship than the randomized controlled intervention study where that had been done?

"A. I don't recall specific instances. But I am aware, I believe, in pharmacoepidemiology and some RTC's of dietary supplements where there have been some issues regarding selection bias and dropout in RCTs where they have not provided the best evidence.

But I think collectively overall at least in theory they are designed to provide the strongest scientific evidence, at least given those parameters I set forth earlier."

THE WITNESS: Yes. So it certainly --

Q. BY MR. METZGER: There's no question.

The next section in this document has a heading of Observational Studies.

Do you see that?

A. I do.

Q. And that section begins with the statement that, "Observational studies measure associations between the substance and disease."

Do you agree with that?

A. I do.

Q. Then it says, "Observational studies lack the controlled setting of intervention studies."

Do you agree?

A. If by controlled setting this refers to an experimental intervention setting, then, yes, observational

studies observe individuals in the natural environment.

Q. Okay. The third sentence in this section says:

"In contrast to intervention studies, observational studies cannot determine whether an observed relationship represents a relationship in which the substance caused a reduction in disease risk or is a coincidence."

Do you agree?

A. Again, in theory, as I've testified to, observational studies provide evidence for or against a hypothesis of association.

Q. Okay. New topic.

You have published a number of meta-analyses regarding particular substances and health outcomes, correct?

A. I have.

Q. For any of those substance/disease relationships which you have investigated and published a meta-analysis, have you concluded causality?

A. I may have indicated that the evidence provides or that the data provide evidence against a conclusion of causality.

Q. When you say --

A. Just like I have here for this matter.

Q. Well, you just answered that you may have. Anything is possible.

Do you actually have a specific recollection or can you direct me to any meta-analysis that you have published where

you actually concluded causality?

A. Again, your use of "concluded causality." I think we're mixing signals here.

I believe there are some papers where I said there was a lack of an independent association, therefore there's no basis for conclusion of causation.

Q. Can you identify any such paper?

A. I would have to look at the results and conclusions of all my publications.

Q. All right. So now that we're on the topic of meta-analysis, just give me one second. I need to find something. Excuse me, your Honor.

Oh, you've got it there. Here we go.

The next exhibit is what? Alex?

MR. INFANTE: 61838.

(Exhibit 61838, Program Schedule, marked for I.D.)

Q. BY MR. METZGER: I'm providing you with Exhibit 61838.

Tell me if you recognize this document, please.

A. I believe I have seen this before, yes.

Q. Okay. So this is a program schedule for a Defense Research Institute seminar for the lawyers at which you spoke, correct?

MR. KENNEDY: Objection, your Honor. Not a document he read, considered or relied on in connection with this case.

THE COURT: Overruled.

THE WITNESS: I believe this is a program, and I believe my name is listed on it.

Q. BY MR. METZGER: Right.

And the title of your presentation to the defense lawyers was Lies, Damn Lies and Statistics: The Use and Limitations of Meta-Analyses in Litigation, correct?

A. I believe that to be the case, yes.

Q. All right. And you actually presented a paper at this conference, did you not?

A. I did.

Q. And what's the next exhibit?

MR. INFANTE: 61839.

(Exhibit 61839, Article, marked for I.D.)

Q. BY MR. METZGER: And you co-authored that paper with Bruce Parker from the law firm of Venable, correct?

A. Yes.

Q. And that paper was titled Meta-Analysis: Recycling Garbage or an Important Tool for Evaluating the Evidence, correct?

A. Yes.

Q. And in the introduction to this article, this paper, you wrote that, "Meta-analysis is a statistical tool that, like any tool found in a hardware store, can be very helpful when used in the right manner, but when misused can make the job more difficult or even damaging," correct?

A. Yes. I did not write that particular sentence, but, yes, that's what's indicated right here.

Q. Well, you read this entire paper and you approved it, didn't you?

A. Yeah, I agree with that statement.

Q. Okay.
A. Yeah.
Q. And the fifth line you wrote -- or this paper that you authored says:
"It should come as no surprise to any defense lawyer that plaintiffs' experts misuse this tool to create associations that don't exist.
The difficulty for the defense lawyer is being able to demonstrate in an understandable manner to a jury that corners have been cut on by the expert performing the meta-analysis and how doing so produced a false result."
Correct?
A. That's what's written.
Q. Turn to page 2, please, the second paragraph.
In the middle of the paragraph, you wrote, "However, the quality of the published meta-analyses is variable."
That's true, isn't it?
A. I'm are so. Where are you?
Q. Page 2, the second paragraph in the middle.
A. Okay.
Q. You agree that the quality of published meta-analysis is variable?
A. Oh, yes.
Q. Yeah.
Then it says here, "Unfortunately a non-trivial

proportion of published meta-analyses convolute interpretation rather than make the scientific evidence clearer."

That's what you wrote, correct?

A. Yes. That's why we need experts such as myself who are well versed in meta-analysis to review them, yes. Absolutely.

Q. All right. Now, turn, if you would -- we're going to move far ahead in this document to page 15.

And there is a new section here. Do you see that, Objectivity versus Subjectivity?

A. I do see that.

Q. And immediately before that, there is a phrase that says, quote, "If poorly conducted meta-analysis" -- I'm sorry.

"If poorly conducted, a meta-analysis may yield a false sense of consistency in the literature."

That's something that you approved, correct?

A. I think we should -- I would like to acknowledge the entire sentence. That's just part of that sentence.

Q. Okay. Well, Mr. Kennedy can take up this whole document with you if he wishes.

All right. So now turn to page 16. And there's a heading which says, "A Meta-Analysis Inherently Examines Study Quality."

Do you see that?

A. Yes.

Q. And you wrote here.

"The value and utility of a

meta-analysis is largely dependent upon the type of information on which it is based, the clarity of methodology and reporting, the quality and comprehensiveness of the systematic process and the interpretation of the literature."

That you wrote, right?

A. Absolutely.

Q. Okay. Then you wrote:

"It is important to consider the methodological quality of studies that are included in a meta-analysis since the results of a meta-analysis are only as valid as the studies included in the model. This has been referred to as the garbage-in/garbage-out phenomenon."

That's what you wrote?

A. Yes.

Q. In the very middle of that paragraph there's a sentence that says, quote:

"If the quality of the studies included in the review are compromised and/or prone to biases, a synthesis of their results will not be able to eliminate these original flaws."

You wrote that, right?

A. Yes, absolutely.

Q. And the last sentence on this page is:

"On the other hand, a meta-analysis of well conducted, randomized controlled clinical trials may produce an accurate and valid summary association and allow for the evaluation of patterns of associations across population subgroups."

You wrote that, correct?

A. Yes. In this particular context and this topic area, absolutely.

Q. All right. You yesterday spoke about so many meta-analyses that you had reviewed for this case. I don't recall the number, but I think it was in the hundreds. Does that seem right?

A. At least.

Q. Would you tell the Court how many of those meta-analyses were meta-analyses of well-conducted, randomized controlled clinical trials evaluating a substance and a disease?

A. Very few.

Because, again, as I said yesterday, it's not the right tool for the trade in this type of topic area.

Q. All right. Now, would you turn to page 24.

You wrote here:

"Rather than using meta-analysis to generate a more precise relative risk, meta-analysis is more likely to be used by defense attorneys than their experts to

demonstrate that the plaintiffs' evidence lacks consistency."

You wrote that, right?

A. I did not write that. I'm not sure exactly where you are.

You said page 24.

Q. At the very top.

You approved of that, correct?

A. I'm terribly sorry. I still am not seeing exactly where you are.

MR. METZGER: May I approach, your Honor.

Q. I'm sorry?

A. You said 24.

Q. I guess when it printed out it's different.

It's at the bottom of your page 23. I don't know what happened here.

A. Okay.

Q. The sentence is:

"Rather than using meta-analysis to generate a more concise relative risk, a meta-analysis a more likely to be used by defense attorneys and their experts to demonstrate that the plaintiffs' evidence lacks consistency."

Did you approve of that?

A. I did. A meta-analysis can be used in -- the purpose of a meta-analysis is to evaluate consistency, absolutely.

Q. And then it says, "This can be accomplished by demonstrating statistical heterogeneity or design heterogeneity."

Did you write that or approve of that?

A. Yes.

Q. It then says:

"If the goal is to demonstrate the unreliability of the plaintiffs' meta-analysis, defense counsel may want to use empirical data suggesting the unreliability of meta-analysis compared to randomized clinical studies."

Did you write or approve that?

A. I'm sorry. I'm just reading it.

I read it, yeah.

The point is, I'm considering all levels of evidence.

Q. Okay. And then you write here, "For example, a paper published in the NEJM" -- that's the New England Journal of Medicine, correct?

A. It is.

Q. (Reading:)

-- "in 1997, discrepancies between meta-analysis and subsequent large randomized controlled trials, 337 New England Journal of Medicine, page 536, compared 19 meta-analyses published on different health issues before a large randomized study had been conducted on the

question. For 40 primary and secondary outcomes predicted by the meta-analyses, there was only fair agreement between the meta-analyses and the gold standard RCT. The authors concluded that had no RCT been conducted, meta-analysis would have suggested treatment in 32 percent of cases that was not found efficacious by an RCT and a rejection of efficacious treatment in 33 percent of the cases."

That's what you noted here, correct?

A. That's what's here.

However, this is talking about treatment in drug trials after diagnosis of disease. So it's not relevant to what I did in this matter.

MR. METZGER: We will we mark as Exhibit 618 -- is this 40 now?

61840 the New England Journal of Medicine article referenced.

(Exhibit 61840, NEJM Article, marked for I.D.)

Q. BY MR. METZGER: Let's look at the conclusion of this article.

This is the article that is referenced, is it not, Dr. Alexander?

The conclusion of the article is:

"The outcomes of the 12 large randomized controlled trials that we studied were not predicted accurately 35 percent of the time

by the meta-analyses published previously on the same topics."

So that's the conclusion, correct?

A. It is, for drug treatments, yes.

Q. All right. So this is a reporting a 35 percent error rate of meta-analyses, is it not?

A. Again, in this specific context of drug treatments, that's what the authors are indicating here.

Q. Right. And in the context of nutritional epidemiology, which is much more confounded, there would be an even higher error rate, would there not?

A. No. You can't draw that conclusion whatsoever.

Q. Okay.

MR. METZGER: Your Honor, would this be an appropriate point for a morning break?

THE COURT: Are you asking for a break?

MR. METZGER: I'm asking for a break.

THE COURT: I mean --

MR. METZGER: What time do you prefer having morning breaks?

THE COURT: Around 10:45.

MR. METZGER: Okay. All right. Then I'll go on to a new topic.

I just need a moment here. Okay.

Q. So let's talk about nutritional epidemiology.

First, is it true that despite billions of research dollars and decades of research, few if any foods have been clearly causally associated with increasing or decreasing the

risk of cancer?

A. Yes. Based on my statement over time.

Q. Okay. I'm showing you Exhibit 61841, a letter by you dated September 8, 2015.

(Exhibit 61841, Letter, marked for I.D.)

Q. BY MR. METZGER: Do you recognize the document?

A. I do.

Q. All right. And so this is a letter that you wrote to the IARC Working Group regarding meat or processed meat and cancer, correct?

A. This was unprocessed red meat and processed meat, yes.

Q. Okay. And I would like to go through the second paragraph of this with you.

You wrote here, "The potential role that red meat or processed meat intake plays on cancer risk has been widely debated in scientific communities."

Do you see that?

A. I do.

Q. Okay. You write, then, "Indeed, interpreting findings from epidemiologic studies of dietary factors such as individual foods or food groups involves numerous methodological considerations."

That's true, is it not?

A. It does, yes.

Q. And then you list what some of these are.

And you write, "Clearly and specifically defining the food variables; i.e., exposure," correct?

A. Yes.
Q. The outcomes of interest?
A. Absolutely.
Q. Accurately measuring food intake?
A. Yes.
Q. And you consider that a foremost challenge in nutritional epidemiology, correct?
A. Yes, it is.
Q. Accounting for dietary pattern differences across populations?
A. Yes.
Q. Understanding the role of bias and confounding within and across studies?
A. Absolutely.
Q. Isolating the effects of a single food or food group from the countless foods and dietary constituents that individuals consume on a daily basis?
A. Yes.
Q. As a matter of fact, you have questioned whether that's even possible, haven't you?
A. Have I questioned whether it's possible?
Q. Yeah.
A. I think it's challenging. It's most definitely challenging and something that we have to consider. That's why we look at the consistency of associations across studies.
Q. And you also point out, "Assessing potential and relevant biological mechanisms and genetic variation in metabolizing enzymes," right?

A. Yes.

Q. And, incidentally, regarding your conclusions for this case, you did not consider biological mechanisms at all, did you?

A. I did not consider or evaluate postulated mechanisms. I focused on the human health epidemiological data.

Q. You also did not consider genetic variation of metabolizing enzymes for your conclusions in this case, right?

A. Correct. I focused on the human health epidemiology.

Q. All right. And also statistical testing parameters, you write here.

Then you write:

"What makes interpretation even more challenging is the fact that prospective cohort studies generate associations between foods and cancer that are very weak in magnitude, with most relative risks ranging between 0.8 and 1.25."

Right?

A. Yes.

Q. And then you write:

"Given the considerable degree of exposure misclassification from self-reported dietary intake, correlation of certain foods with other dietary and lifestyle factors and the impact of bias and

confounding, there is significant uncertainty surrounding the epidemiologic evidence for foods and cancer."

That's what you wrote?

A. Yes. For foods and cancer, yes.

Q. And you would also include beverages such as coffee within that context of foods, correct?

A. Well, I think, of course, it's a -- coffee, foods, beverages and cancer, it's a challenging undertaking. That's why we need the systematic approach that I took.

Q. All right. Then you conclude:

"In fact, despite billions of research dollars and decades of research, few if any foods have been clearly causally associated with increasing or decreasing the risk of cancer."

Incidentally, you haven't even questioned whether there's a causal relationship between consumption of fruits and vegetables and cancer, right?

A. I have even questioned?

Q. Yes.

A. I think it's a very common research topic.

I think many researchers have questioned that relationship for certain types of cancer.

MR. METZGER: Okay. We will mark as the next, which is 61842.

(Exhibit 61842, Letter, marked for I.D.)

Q. BY MR. METZGER: Another letter of yours, this

one to Dr. Lunn.

Who is Dr. Lunn, by the way?

A. I'm sorry, who?

Q. Dr. Lunn, L-U-N-N.

A. I'll have to see after you provide it to me.

Q. Sure. Here you go.

You do recognize Exhibit 61842 as a letter you wrote, correct?

MR. KENNEDY: Your Honor, we'll object. As far as we know, this wasn't on any of the exhibit lists.

Maybe Mr. Metzger can identify where this was produced.

MR. METZGER: I'll identify it as impeachment.

THE COURT: Objection overruled.

THE WITNESS: The letter looks familiar. I don't recall specifically who Dr. Lunn is.

Q. BY MR. METZGER: Well, the letter is a letter that you wrote, correct?

A. It appears to be one I've written.

Q. To help you identify Dr. Lunn, if you look at the bottom of the first page, you write that, "I kindly ask that you earnestly consider my forthcoming scientific comments to the Office of the Report on Carcinogens in response to its September 9, 2016 Federal Register."

So Dr. Lunn is with the Office of the Report on Carcinogens, a governmental agency, correct?

A. I see, yes.

Q. All right. So I would like you to turn now to the third page of this document, the letter you wrote.

And the first -- I'm sorry, the second sentence I would like to direct your attention to.

You wrote, quote:

"The interdependency of food consumption with other dietary and lifestyle factors, socioeconomic characteristics, clinical variables and genetic traits makes it difficult to isolate the independent effects of a specific food or food group such as meat intake on disease risk."

That's what you wrote?

A. Yes.

Q. And the same would apply for coffee, would it not?

A. It's a similar situation.

Q. Right.

A. Which is why we undertake this type of approach, yes.

Q. And then you write, quote:

"Interpretation of findings from nutritional epidemiology studies are further complicated by the fact that this research area is particularly prone to reporting bias because of the numerous types of foods, food combinations, nutrients and cooking methods ascertained on a typical food frequency questionnaire."

Right?

A. It does. It depends on the type of food and the outcome regarding the reporting bias aspect.

Q. Right.

A. But it is variable.

Q. Right.

As a matter of fact, being very familiar with food frequency questionnaires for the diet and cancer, it is your belief that those studies that use food frequency questionnaires should not be viewed as a good measuring stick for reliability, true?

A. I'm sorry.

You're reading this from somewhere? Or was that --

Q. I'm asking you a question.

Is that true?

A. I'm sorry. Because you were reading before so I didn't know if I was supposed to find something on the paper.

Q. It's not on the document there. I'm asking you a new question. I apologize.

A. I'm sorry. Can you repeat that, please?

Q. I apologize for the poor transition.

So my question is that, being very familiar with food frequency questionnaires for the diet and cancer, you believe that epidemiologic studies using food frequency questionnaires should not be viewed as a good measuring stick for reliablity, true?

A. Not necessarily. It certainly depends on the scientific topic area and what we're evaluating.

MR. METZGER: All right. So we'll now mark as

Exhibit 61843 some testimony that you gave to the U.S. EPA.

(Exhibit 61843, Document, marked for I.D.)

Q. BY MR. METZGER: So this document is entitled "United States Environmental Protection Agency, Federal Insecticide, Fungicide and Rodenticide Act, Scientific Advisory Panel: Open Meeting to consider and review draft framework in case studies on atrazine, human incidence and the agricultural health study, incorporation of epidemiology and human incident data into human health risk assessment," dated February 2, 2010.

This is a U.S. EPA meeting that was -- you spoke at, correct?

A. I believe I did. I don't recall ever seeing this specific document, though.

Q. Well, would you turn to -- let's see.

If you look at what's page 282 of the transcript, the third page of the document, the chair of this meeting says, "I'm going to move ahead with the next public commentator or presenter, and that will be Dr. Dominik Alexander, representing Exponent."

So this was at the time you were with Exponent, correct?

A. It would have been.

Q. Right. And would you turn to page 298 of this document. It's toward the end.

If you look at lines 5 through 9, what you told the United States EPA was that, quote:

"Doing a lot of work in nutritional

epidemiology and being very familiar with the food frequency questionnaire for diet and cancer, those studies should not be viewed as a good measuring stick for reliability."

That's what you told the United States EPA, correct?

A. I believe that's out of context. That's for a very specific situation.

Q. Okay. Dr. Alexander, are you aware of any international organization or governmental authority that has actually concluded that coffee consumption prevents any disease?

A. I'm not aware.

Q. Have you read any published peer-reviewed article in a reputable journal that has concluded that coffee consumption actually prevents any type of cancer?

A. I don't know if they've indicated "prevents." Certainly numerous indicate decreased risks.

Q. Okay. Are you aware of any international or governmental organization or agency that has concluded that consumption of coffee causally prevents the development of any chronic disease or cancer?

A. Same response. I don't recall "causally prevents," but they certainly do indicate decreased risk.

Q. Right.

A while ago I asked you -- I'm not sure if I asked you this question.

Have you ever questioned whether it is possible to

isolate an individual food component to determine causality for that food component?

A. You will have to clarify that for me, please.

Q. Well, can I just read your answer from the deposition -- you seemed to understand it at the deposition.

A. Well, it's a question taken in isolation, so I'm not sure what led up to it or what followed it.

THE COURT: All right. Just read the deposition.

MR. METZGER: The deposition, page 293, line 24, through page 294, line 3.

MR. KENNEDY: Your Honor, could I have a second?

THE COURT: Yes.

MR. METZGER: I apologize.

MR. KENNEDY: I don't think it's impeaching.

No other objection, your Honor.

THE COURT: All right. Thank you.

MR. METZGER: (Reading:)

"Q. Okay. Have you ever questioned whether it is possible to isolate an individual food component to determine causality for that food component?

"A. I have questioned the ability to independently isolate an individual food item. Yes."

Q. Okay. Dr. Alexander, we've been talking about association and causation.

There is a big distinction between association and causation, isn't there?

A. I think they are relatable concepts. I wouldn't necessarily call it a big distinction. It depends on the application of association to a causal framework.

Q. Well, do you recall giving a deposition in the case of Burnett versus Bennett Auto Supply, August 4, 2014?

A. I know I have. I don't recall the specific nature of that matter.

Q. Okay.

A. I remember the name.

Q. All right. I'm going to read from that deposition page 59, line 22, through 60, line 11.

I can provide your Honor with a copy of the deposition.

THE COURT: All right. Please give it to the clerk.

MR. METZGER: Any objection?

MR. KENNEDY: No, your Honor.

MR. METZGER: (Reading:)

"Q. And you did a meta-analysis to determine whether occupational exposure to that substance" -- referring to TCE -- "can cause non-Hodgkin's lymphoma, correct?

"A. No. Well, there's a big distinction between association and causation. So first of all, we take a look -- when we go into a meta-analysis, we look at the associations and then, based on, you know, depending on the nature of the topic, the volume of the literature, the

strengths and limitations, then we can go down the road of causation."

A. Yes. Like I said, it's a relatable concept, and it depends on the application of the situation.

Q. Associations either indicate an increased risk of disease or a decreased risk of disease, correct?

A. They may, yes.

Q. An association indicating a decreased risk of disease is not a health benefit unless the association is causal, true?

A. I've heard it described that way. I think it can provide a framework. Decreased risk indicates that there may be a health benefit, but it's an association as a decreased risk.

Q. And since you have no opinions in this case on causation of health effects from consumption of coffee, you do not conclude that coffee consumption causes any health benefit, true?

A. I am not making a conclusion of causation regarding a health benefit.

Q. Okay.

MR. METZGER: Your Honor, would now be an appropriate time?

THE COURT: Let's take a recess at this time.

We'll be in recess for 15 minutes.

(Recess.)

THE COURT: All right. Back in the trial of CERT versus Starbucks.

Dr. Alexander is on the stand and Mr. Metzger is inquiring.

Counsel, you may proceed.

MR. METZGER: Thank you, your Honor.

Q. Dr. Alexander, I'm looking at slide eight of your demonstrative.

That's the slide where you list six diseases under the heading Independently Associated with Decreased Risk.

Are you with me?

A. I am.

Q. All right. So using your framework of independently associated, you came up with all of the diseases that you evaluated for coffee consumption; these six that you believe are independently associated with decreased risk, correct?

A. Yes. I believe there's sufficient epidemiologic evidence to support a conclusion of an independent decreased risk.

Q. Right. And one of them is liver cancer, correct?

A. Yes.

Q. All right. And your conclusions were based -- regarding liver cancer in relationship to coffee consumption was based upon meta-analyses, correct?

A. Meta-analyses as well as the individual studies, with the understanding that meta-analyses reflect the weight of evidence from the individually conducted studies.

Q. Okay. I'm going to show you Exhibit 57649,

meta-analysis by Kennedy, et al., published in the British Medical Journal, "Coffee, including caffeinated and decaffeinated coffee, and the risk of hepatocellular carcinoma, a systematic review and dose-response meta-analysis," published in 2017.

This is one of the meta-analyses, perhaps the most recent meta-analysis, regarding coffee consumption and liver cancer that you reviewed, correct?

A. I don't recall if it's the most recent. There is one that's even more recent.

Q. Okay.

A. But it's 2017.

Again, there is two of them.

Q. All right. So this is one that you have reviewed, though?

A. Yes.

Q. Okay. And just so it's clear, where it says on the title hepatocellular carcinoma, that's liver cancer, right?

A. It is.

Q. Okay. Would you take a look at page 11 of this study.

And by the way, this study, like the other meta-analyses for liver cancer, reported a significantly decreased risk -- statistically, correct?

A. A statistically significantly decreased risk, yes.

Q. As an association?

A. Well, yes, that's what a statistical significance represents, yes.

Q. And I think you call it -- because it's a decreased risk, this is what we would call an inverse association, correct?

A. Yes, that is correct.

Q. All right. So now looking at page 11, if you look at the second full paragraph, right in the middle of the page, there is a sentence that says, "The main limitation is that all the included studies were observational."

Let me stop there.

So what they're talking about here by included studies, those are the individual epidemiologic studies which they included in the meta-analysis to derive a meta risk; is that correct?

A. Yes. The observational studies were included in this meta-analysis.

Q. Yeah. So this is not a meta-analysis of randomized control trials. This is a meta-analysis of observational epidemiologic studies, correct?

A. Correct. Because obviously it wouldn't make sense to use an RCT for liver cancer and coffee. So, correct.

Q. So where it says here, "The main limitation is that all the included studies were observational, and thus we cannot infer causation," do you see that?

A. I see where you're reading from.

Q. So the authors of this very recent meta-analysis of coffee consumption, both caffeinated and decaffeinated, in

liver cancer concluded that because this meta-analysis was just based on observational epidemiological studies, they could not conclude causation, correct?

A. That's what they say there, but they do use the word "protective" in their conclusions.

Q. All right. Thank you.

Now, I understand -- I'm assuming, based upon correspondence I received from counsel, that after you gave your deposition you did some more work in this case; is that correct?

A. I did.

Q. All right. And are you aware that after your deposition some of the experts that were retained by my office gave their depositions?

A. Yes.

Q. And that they provided written summaries of their opinions?

A. Yes.

Q. All right. And did you receive those?

A. I did.

Q. Okay.

A. At least for some.

Q. Okay. So let me -- and you reviewed those?

A. I did for the ones that I received, yes.

Q. And did you also review the studies that were referenced in those summaries of those experts' opinions?

A. I did review those studies.

Q. You made a substantial effort to read all those

studies that the plaintiff's experts were relying on?

A. I reviewed studies that they cited, some of which were studies that I had cited in my review, as well.

So there's a considerable amount of overlap.

Q. Okay. So I'm going to give you what's been marked as Exhibit 59967. The opinions of Jack James.

Is this one of the sets of opinions that you reviewed?

A. I believe it is.

Q. Okay. And if you turn to page 4, Dr. James provided opinions regarding pregnancy outcomes.

Do you see that?

A. Yes, on page 4?

Q. Right. And he cited apparently four of his own articles as materials he was relying on.

Do you see that?

A. I do.

Q. Okay. Did you happen to read his articles?

A. I did look at his articles once I received them.

Q. Okay. So under pregnancy outcomes, let's move ahead to page 6 of these opinions where he is addressing outcomes.

One of the outcomes that he addresses is reduced fetal weight and growth, correct?

A. Yes.

Q. And another one that he identified is pregnancy loss, including spontaneous abortion and stillbirth, correct?

A. On the next page, yes.

Q. Correct. Okay. Now, regarding these -- by the

way, these are all outcomes that concern maternal consumption of coffee during pregnancy and outcomes to the fetus or the child, correct?

A. That's my understanding.

Q. All right. And do you note here that Dr. James had identified several meta-analyses regarding the effects of maternal consumption of coffee or caffeine during pregnancy and reproductive developmental outcomes?

A. I see some meta-analyses cited by Dr. James.

Q. And did you read those meta-analyses?

A. I would have, yes. Yes.

Q. All right. So let's look under the section on page 6 regarding reduced fetal weight and growth.

There is a meta-analysis by Fernandez, 1998, do you see that?

A. I do.

Q. And that's one you reviewed, correct?

A. Yes.

Q. And there's one by Santos, 1998, which you also reviewed?

A. Yes.

Q. And one by Chen, 2014, which you reviewed?

A. Yes.

Q. And one by Greenwood, 2014, which you reviewed?

A. Yes.

Q. And one by Rhee, R-H-E-E, 2015, which you reviewed?

A. Yes.

Q. So there are five meta-analyses here that Dr. James considered regarding reduced fetal weight and growth.

Each of these meta-analyses that Dr. James referenced here reported significantly increased risks of low birth weight from maternal consumption of coffee during pregnancy, true?

A. They may have. I would have to take a look at all the different analyses within those studies, but I do know that some did, yes.

Q. Well, I want to be sure that -- here it is.

So I'm going to provide you with each of these so we can just briefly -- so you can have them and look at them.

So Exhibit 51101 is the Fernandez meta-analysis.

Here you go.

A. Thank you.

Q. Exhibit -- let's see -- 59449 is the Santos meta-analysis, okay?

And let's see. Exhibit 56276 is the Greenwood meta-analysis.

Exhibit 55439 is the Rhee meta-analysis.

Those are the five.

Do you have them all now?

A. Are two stapled together?

Q. Is there a mistake in the copying?

A. I believe I have four. This one seems thick.

Q. Wait a second. Why do you only have four? What am I missing?

Oh, I'm sorry. I forgot the Chen meta-analysis.

This one has your Bates number on it so it's one that you originally had for your deposition, but I don't have an exhibit number on it.

MR. METZGER: So what's the next exhibit?

MR. INFANTE: 61844.

MR. METZGER: 61844.

(Exhibit 61844, Chen Meta-Analysis, marked for I.D.)

Q. All right. Here is the Chen meta-analysis.

Now I think you have all five, correct?

A. Okay. I should.

Q. So if you just look at the abstracts, you will be able to answer the question that I want to ask.

Can you confirm for the Court that each of these meta-analyses that we've just identified reported significantly increased risks of low birth weight from maternal consumption of coffee during pregnancy?

A. Looking at the abstracts, I can't.

For example, in Santos, no effect of caffeine on low birth weight. Results did not change after control for confounders. It doesn't have data.

Fernandez, I believe some. So I don't think the -- at least in all of these the abstract is indicating that.

Q. Well, you're referring to the Fernandez abstract. It says the overall risk ratio was 1.51.

A. By Santos. I'm sorry. I'm not sure if I said Fernandez.

Q. Santos.

So let's take them one by one, then.

Can you confirm that the Fernandez meta-analysis reported a significantly increased risk of reduced fetal growth or weight from maternal consumption of coffee or caffeine during pregnancy?

A. They did.

However, they also say control for confounders such as age, smoking and ethanol was not possible.

Q. Okay.

A. So methodological limitations notwithstanding, yes.

Q. Regarding methodological limitations, all observational epidemiologic studies have methodological limitations, don't they?

A. All studies have potential for methodological limitations. That's why we need to evaluate study quality and the parameters of each evaluation.

Q. Right.

It's not just the studies that report adverse -- increased risks of adverse effects that have methodological limitations. It's also those that report decreased risks of effects, true?

A. Of course.

Q. Okay. All right. So that's Fernandez.

Can you confirm that in the Chen meta-analysis they also reported a significantly increased risk of reduced fetal weight or growth from maternal consumption of coffee or caffeine during pregnancy?

A. I'm sorry. I'm just looking for fetal growth. I don't think they have -- you said Chen, correct?

Q. Uh-huh.

A. I don't think they have fetal growth in the abstract unless I'm reading this incorrectly.

Q. (Reading:)

"In the dose-response analysis, each 100-mg/day increment in maternal caffeine intake (around one cup of coffee) was associated with 13 % higher risk of low birth weight (relative risk 1.13), 95 percent confidence interval (1.06 to 1.21)."

That's significant, isn't it?

A. I'm sorry. Where are you reading from? That's not the abstract; is that correct?

Q. Yeah. I guess I'm reading actually from Dr. James' summary of it.

Can you confirm that that's correct?

A. It's not in the abstract. It may be in the body of the article.

Actually, I don't see that listed in terms of the results here.

I believe that the authors looked at risk of pregnancy loss. I don't think that the authors looked at growth.

Q. Well, okay. So if you're looking at pregnancy loss, was that significantly increased, the risk?

A. So not growth but loss.

There is a subgroup or analysis with that, yes.

Q. Okay. So that's the Chen study.

What about the Greenwood study? Did that likewise report a significantly increased risk of pregnancy loss from maternal consumption of coffee or caffeine during pregnancy?

A. Okay. So now we're on pregnancy loss, then?

Q. Yes, which would be -- that would include both spontaneous abortion and stillbirth, correct?

A. Yes.

So stillbirth is not significantly associated here.

Spontaneous abortion, based on the abstract, there's a small increase in risk.

Q. Okay. And this one also shows decreased birth weight, correct?

A. Yes, a small, small increase in risk, yes.

Q. All right. And also pre-term delivery?

A. Let's see. I may be reading this wrong.

Pre-term delivery, that's the one that is not statistically significant.

Q. All right. So let's go on to the Rhee meta-analysis, the most recent one, 2015.

That one also reports adverse reproductive developmental effects from consumption of coffee or caffeine during pregnancy, true?

A. The Rhee manuscript looks at low birth weight.

Q. Okay. And that was significantly increased, correct?

A. Yes, they did have a finding of significance.

Q. And also -- okay. So there was also a study, a meta-analysis, by -- let's see.

There was also a meta-analysis by Li, L-I, a meta-analysis of pregnancy -- of risk of pregnancy loss with caffeine and coffee consumption during pregnancy, which will be the next exhibit.

MR. METZGER: Which is what number?

MR. PARISER: 61845.

MR. METZGER: 61845.

(Exhibit 61845, Li Meta-Analysis, marked for I.D.)

Q. And this is another meta-analysis that Dr. James cited that you reviewed, correct?

A. Yes.

Q. And this meta-analysis reported significantly increased risk of pregnancy loss from caffeine and coffee consumption during pregnancy, correct?

A. Yes. A small increase that's significant, yes.

Q. Okay. And I see. That's why I was confused.

MR. METZGER: I'm going to mark as 61846 another meta-analysis, a different one by Chen.

(Exhibit 61846, Chen 2014 Meta-Analysis, marked for I.D.)

Q. BY MR. METZGER: That's why you weren't finding it. 61846. This one published in 2014.

And is this another meta-analysis cited by Dr. James that you reviewed?

A. Yes.

Q. And the title is "Maternal caffeine intake

during pregnancy is associated with low birth weight: a systematic review and dose-response meta-analysis."

That describes what the finding was, correct?

A. That's what the title is.

Q. And if you look at the abstract, you'll see that, do you not?

A. I do. In the results.

Q. All right. So we have here several meta-analyses that have reported significantly increased risks of adverse effects to the newborn from maternal consumption of coffee or caffeine during pregnancy, correct?

A. We've talked about low birth weight and I believe pregnancy loss.

Q. Okay. Now, in your opinions I did not note that you cited any meta-analyses that reported increased risks of disease. Were there any?

A. Cited where?

I would have cited all of these studies.

Q. Well, I'm sorry.

I'm looking in the binder that was provided regarding your opinions, the demonstrative.

Do you have that binder?

A. I do have the binder, yes.

Q. So if you would look through the demonstratives that you prepared, there aren't any meta-analyses there that you reported or that you cited as reporting increased risk of disease; is that true?

A. No.

Q. What meta-analyses did you cite that report increased risk of disease?

A. I didn't cite specifically meta-analyses.

What this is is a summary of the associations.

So I've cited end points in diseases for which some meta-analyses may indicate a increased risk. Some may indicate a decreased risk.

So I wasn't citing them based on meta-analyses of associations. These were based on the summary of evidence, more than just the meta-analyses findings.

Q. I guess I'm a little puzzled because for this case you have not considered mechanistic issues or animal studies or in vitro or in vivo data that go into a causal analysis. You've only been considering the epidemiologic studies, right, and the meta-analysis of them?

A. Yes. I think we're on different pages here.

I think I'm misreading what you're asking, perhaps, and I think perhaps you're misreading what I'm doing for my systematic approach in the meta-analysis.

Q. Would you take a look at the document that counsel has requested be, I guess, admitted into evidence, which is 73528, the one that says "No independent association." It has three columns.

A. Yes.

Q. Okay. And you have here -- I think it was counted to be 30 outcomes, health outcomes, which you have assessed for association, correct, or independent association?

A. I've assessed on the basis of whether the

evidence supports a conclusion for an independent association.

Q. Correct. And did you consider meta-analyses in reaching those conclusions, in listing these 30 outcomes?

A. I did as a basis for evaluating the state of the epidemiologic science. Yes.

Q. Okay. And were there any meta-analyses that you considered regarding these 30 outcomes that reported significantly increased risks of the outcome?

A. Yes.

Q. For which diseases or outcomes?

A. I believe there's meta-analyses of lung cancer, I believe of stomach cancer. There may be some -- one of fracture.

Q. Of fractures, is that bone fracture?

A. Yes.

Q. Uh-huh.

A. There may be different subgroups, for example, case-control studies for pancreatic cancer, so, certainly.

Q. And there were also meta-analyses of coffee consumption in bladder cancer that reported significantly increased risks, true?

A. Yes.

Q. All right. And some even reported monotonic dose-response relationships, correct?

A. Some individual studies.

Q. Meta-analyses do that, right?

A. Yes, meta-analyses, yes.

Q. Okay.

A. Particularly for case-control studies.

Q. Okay. So bone fracture is another one for which there was a meta-analysis that reported significantly increased risks from coffee consumption, correct?

A. I believe so.

Q. Right. And there's -- okay.

Your Honor, I apologize if this is taking a little bit long, but I'm promising you that this is going to reduce the number of plaintiff's experts that are going to have to testify.

THE COURT: All right. We're going to hold you to your promise.

Q. BY MR. METZGER: If we look at your list, you have on Exhibit 73528 -- do you have childhood leukemia on this list?

A. I don't know if I have that exhibit number. It may be on the second exhibit.

Q. Oh, I see.

You have childhood leukemia for limited and insufficient evidence, correct?

A. Yes.

Q. Okay. There are a number of meta-analyses that have been published regarding maternal consumption of coffee during pregnancy and childhood leukemia, true?

A. I believe a few, based on the case-control studies.

Q. I'll provide you Exhibit 51781, a meta-analysis on that topic by Chang.

And you're familiar with that, correct?

A. Yes.

Q. And I'll provide you a meta-analysis by Thomopoulos, for which we need an exhibit number.

This is one which you produced.

MR. PARISER: 61847.

MR. METZGER: 61847.

(Exhibit 61847, Thomopoulos Meta-Analysis, marked for I.D.)

Q. BY MR. METZGER: And this is another meta-analysis that you reviewed regarding childhood leukemia, correct?

A. Yes.

Q. And then there was -- I'm not seeing it here. There was another one by Yan. Do you recall that?

A. I do.

Q. And each of these three meta-analyses regarding consumption of coffee during pregnancy and childhood leukemia reported significantly increased risks of childhood leukemia from maternal consumption, true?

A. They did report an increased risk, yes.

Q. Right. And they all, as you point out, case-control studies, correct?

A. They are.

Q. And there is a reason for that, isn't there?

A. There may be. Cohort studies can certainly be done there, maybe.

Q. Well, actually, to do a cohort study for

maternal consumption of coffee and childhood leukemia, you would need a huge population just to get enough cases of childhood leukemia to be able to do any statistics, wouldn't you?

A. You may need a large sample size, but Chang, in one of the meta-analyses that you handed me, in their conclusion they said prospective studies are needed.

Q. Of course they're needed. And so are randomized controlled trials, aren't they?

A. They're not applicable. It's not the right tool for the trade.

Q. I see.

So because there have been no randomized controlled trials done for coffee consumption and chronic disease or cancer outcomes, it's not the tool of the trade. But it's all right to disregard case-control studies for a rare outcome like childhood leukemia when that is the tool of the trade, right?

A. I think that mischaracterizes it.

For those other outcomes we have a very large and robust volume of prospective cohort studies that are well defined. In this body of literature we have eight case-control studies with methodological limitations.

Q. All right. Gastric cancer. There is three meta-analyses regarding coffee consumption and gastric cancer, true?

A. There may be more. I believe there are more.

Q. Okay.

A. There are several. That's one of the most widely studied areas in nutrition.

Q. Okay. Well, all of the meta-analyses regarding coffee consumption and gastric cancer report significantly increased risk, do they not?

A. No, they don't.

Q. Which are the meta-analyses regarding coffee consumption and gastric cancer that you're aware of?

A. Would you like me to read some off?

Q. Just the authors.

A. Okay. There are many associations that are null after --

Q. Just the authors. Just identify the authors.

A. Wang. I don't know how to pronounce this, Xie, X-I-E, Deng, Fang, Li, Sang , Liu, Shen -- that's with an S -- and X-I-E again. I apologize. I don't know how to pronounce that.

Q. One of them you mention is Deng, D-E-N-G?

A. Yes.

Q. That was a meta-analysis of prospective cohort studies, correct?

A. I believe so.

Q. And in the Deng study, the authors found --

A. I do just want to point out, there are more as well. I mean, these were going back to 2014. There are more --

Q. Okay.

A. -- meta-analysis for gastric cancer.

Q. Well, let's look at the this one, the Deng one. This is from 2016, correct?

MR. KENNEDY: Objection, your Honor. We don't have an exhibit number on this one.

MR. METZGER: 61848.

(Exhibit 61848, Deng Meta-Analysis, marked for I.D.)

MR. KENNEDY: 61848?

MR. METZGER: Yes.

THE WITNESS: Deng, yes, I'm sorry. What was the question?

Q. BY MR. METZGER: And in this study, this, meta-analysis, these authors found -- in meta-analyzing the prospective cohort studies, they found a significantly increased risk of gastric-cardia cancer in coffee consumption, a 50 percent increase in risk.

That was significant, correct, if you look at the abstract? It's right there.

A. For gastric-cardia cancer, a specific subgroup in this particular model.

Q. Right.

A. But I recall some mathematic mistakes in this analysis as well.

Q. Okay. And I think you mentioned Shen as another meta-analysis for gastric cancer?

A. Yes.

Q. All right. And that will be 61849, another meta-analysis that you actually had produced for your deposition that has your Bates number on it.

(Exhibit 61849, Shen Meta-Analysis, marked for I.D.)

Q. BY MR. METZGER: And this is another meta-analysis for coffee consumption and gastric cancer dated 2015, correct?

A. It is, but this is an incomplete assessment. They missed several studies.

Q. Okay. Well, what they found was a pool of relative risk of 1.24. That was statistically significant, essentially a 24 percent increased risk, correct?

A. Based on their poorly conducted analysis.

Again, they're missing -- they're missing relevant data points here.

Q. Okay. There is another one you produced at your deposition, 61850, which is Liu, L-I-U, which is 2015.

(Exhibit 61850, Liu Meta-Analysis, marked for I.D.)

Q. BY MR. METZGER: And this one reported a significantly increased risk of cardia -- gastric-cardia cancer, a 23 increased risk, correct?

A. That's what they wrote, but, again, this study also missed some relevant data out there.

Q. All right. By the way, have you read the Guenther study that was recently published?

A. You'll have to be more specific.

Q. Okay.

A. Same outcome?

Q. No, no, no. I'm changing topics here.

We'll get to that later.

It was the recent study. I think it was done by IARC,

a very large prospective cohort study.

A. On -- I'm sorry.

Q. We'll get there. We'll take it up later.

A. Okay. It would be easier if you could show me.

Q. We'll take it up later.

So there was also a meta-analysis done of coffee consumption and rheumatoid arthritis, correct?

A. Yes.

Q. That's one that you produced at your deposition.

MR. METZGER: We will mark 61851.

(Exhibit 61851, Arthritis Meta-Analysis, marked for I.D.)

Q. BY MR. METZGER: And this meta-analysis reported a significant increased risk of rheumatoid arthritis from consumption of coffee, correct?

A. It depends on the model that's being evaluated.

I believe there was an error in this paper and an erratum issued. I believe there's a mathematical error.

Q. Right. And an errata was actually published, which will be Exhibit 61852.

(Exhibit 61852, Erratum, marked for I.D.)

Q. BY MR. METZGER: And that errata corrected that error, did it not?

A. I believe they addressed it, and I believe there is variability in the result here.

Q. Okay. Now, regarding bone fractures, there were a few meta-analyses that were published regarding coffee consumption, true?

A. I believe so.

Q. Okay. One of them was in 2012 by Liu, L-I-U.

MR. METZGER: And that will be 61853.

(Exhibit 61853, Liu 2012 Meta-Analysis, marked for I.D.)

Q. BY MR. METZGER: That's one you've seen, right?

A. Yes.

Q. And this reported significantly increased risk of fracture, bone fracture, from consumption of coffee, correct?

A. It depends on which analytical model that you're looking at.

There were a couple significant findings.

The authors suggested a cautious interpretation because of confounding, but there are a few models that were statistically significant.

Q. Well, this was a meta-analysis of ten prospective cohort studies of over 200,000 participants, correct?

A. Yes.

Q. And there was an overall 3.5 percent higher fracture risk for an increment of one cup of coffee per day, which was significant, correct?

A. In that dose-response model, yes.

However, the authors urged caution for confounding as well as publication bias, which is a concern in this particular study.

Q. Okay. And then there's another meta-analysis

regarding fracture that you reviewed and produced at your deposition which is by Li, L-I, in 2015 entitled "Effect of coffee intake on hip fracture: A meta-analysis of prospective cohort studies."

That is another one that you reviewed, correct?

A. Yes.

Q. Okay.

MR. KENNEDY: Your Honor, could we get the exhibit number on this one?

MR. METZGER: 61854.

(Exhibit 61854, Li 2015 Meta-Analysis, marked for I.D.)

Q. BY MR. METZGER: And then there was another study by Li, L-I, "Coffee consumption and hip risk -- hip fracture risk: A meta-analysis," which will be 61855.

(Exhibit 61855, Li Meta-Analysis, marked for I.D.)

Q. BY MR. METZGER: Which you produced at your deposition.

This is another one that you reviewed, correct?

A. Yes.

Q. Okay. And the pooled odds ratio displayed an increased risk of hip fracture by the 29.7 percent for the highest compared to the lowest coffee consumption, which was not quite statistically significant, correct?

A. Which L-I, which Li study are you on?

Q. Exhibit 61855.

A. Okay. I see where you're reading from, not significant.

And then a more recent other paper by Li reported a 1.13 that was not statistically significant.

Q. Okay. All right.

And then there's another Lee paper, but this one is spelled L-E-E, which will be 61856.

(Exhibit 61856, Lee 2014 Meta-Analysis, marked for I.D.)

Q. BY MR. METZGER: From 2014, entitled "Coffee consumption and risk of fractures: A systematic review and dose-response meta-analysis."

Okay. And in this study the authors found -- they estimated a relative risk of fractures at the highest level of coffee consumption of 1.14, which was statistically significant in women, correct?

A. In women. I see where you're reading.

Q. All right.

A. And statistically significant, inverse, for men.

Q. And in the dose response analysis, the pooled relative risk of fractures in women who consumed two to eight cups of coffee per day were both significantly increased, correct?

A. I see where you're reading from. That's what it says.

Q. So the studies, there are meta-analyses reporting increased risk of chronic diseases from coffee consumption, correct?

A. There are some and there are some subgroups.

And as I testified to yesterday, there are some

relative risks, about 1.0 and below 1.0, which is why we have to consider the totality of these findings and the strength of the evidence.

Q. Right. Okay.

THE COURT: All right. At this time we're going to take our noontime recess.

We'll be in recess until 1:30, at which time we'll resume the testimony of Dr. Alexander.

Thank you, counsel.

(At 12:00 noon, a recess was taken until 1:30 p.m. of the same day.)

SUPERIOR COURT OF THE STATE OF CALIFORNIA

FOR THE COUNTY OF LOS ANGELES

DEPARTMENT 323 HON. ELIHU M. BERLE, JUDGE

| | |
|---|---|
| CERT,<br><br>Plaintiff,<br><br>vs.<br><br>STARBUCKS CORP, ET AL.,<br><br>Defendants. | SUPERIOR COURT<br>CASE NO. BC 435759<br>BC 461182 |

I, DAVID A. SALYER, Official Pro Tem Reporter of the Superior Court of the State of California, for the County of Los Angeles, do hereby certify that the foregoing pages, 1 through 81, inclusive, comprise a true and correct transcript of the proceedings taken in the above-entitled matter reported by me on September 7, 2017.

DATED: September 7, 2017.

DAVID A. SALYER, CSR, RMR, CRR
Official Pro Tem Court Reporter
CSR No. 4410

# EXHIBIT “B”

**Dominik D. Alexander, PhD, MSPH**
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## EDUCATION

| | |
|---|---|
| 2004 | Ph.D., Epidemiology, University of Alabama-Birmingham School of Public Health |
| 2001 | M.S.P.H., Epidemiology and Biostatistics, University of South Florida College of Public Health |
| 1997 | B.A.S., Community Public Health, University of Minnesota |

## EMPLOYMENT

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|---|---|
| 2014–Present | Principal Epidemiologist, EpidStat Institute, Ann Arbor, Michigan. |
| 2004–2014 | Principal Epidemiologist, Exponent Inc. Health Sciences, Chicago, Illinois and Boulder, Colorado. |
| 2001–2004 | Research Assistant, University of Alabama-Birmingham, National Cancer Institute Cancer Prevention and Control Fellowship, Birmingham, Alabama. |
| 2000–2001 | Research Assistant, Moffitt Cancer Center, Department of Radiology, Digital Medical Imaging Program, Tampa, Florida. |
| 2000–2001 | Teaching Assistant, Advanced Epidemiology Methods, Department of Epidemiology and Biostatistics, University of South Florida |

## ACADEMIC APPOINTMENTS

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| 2016–Present | Visiting Professor, Department of Nutrition, Exercise and Sports, Faculty of Science, University of Copenhagen, Denmark |

## HONORS AND AWARDS

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| 2016 | Most read article 2015: *Red Meat and Colorectal Cancer: A Quantitative Update on the State of the Epidemiologic Science*; Journal of the American College of Nutrition |
| 2015 | Appointed to the editorial board of the American Journal of Clinical Nutrition |
| 2013 | Certificate of Achievement, Decker Communication Training |

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| 2010 | UAB School of Public Health Alumnus Award for Scientific Excellence |
| 2010 | MDLinx Featured Article |
| 2001–2004 | National Cancer Institute Cancer Fellowship, Cancer Prevention and Control Training Program, University of Alabama-Birmingham |
| 2003 | William C. Bailey Award for Excellence in Cancer Prevention and Control Research, UAB Comprehensive Cancer Center Annual Research Retreat |
| 2002 | Lifetime Member of MENSA High Intelligence Society |
| 2000–2001 | Academic Fellowship, University of South Florida |

## PROFESSIONAL ORGANIZATIONS

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|---|---|
| 2009–Present | American Society of Nutrition (ASN) |
| 2005–Present | Society for Epidemiologic Research (SER) |
| 2003–Present | American College of Epidemiology (ACE) |
| 2011–2013 | International Society of Pharmacoepidemiology (ISPE) |
| 2005–2008 | International Society for Environmental Epidemiology (ISEE) |
| 2005–2008 | American Public Health Association (APHA) |
| 1999–2001 | Infectious Disease Association (IDSA) |

## PRIMARY AREAS OF EXPERTISE

Meta-analysis methodology

Systematic reviews and weight-of-evidence assessments

Disease causation assessments

Occupational and environmental epidemiology

Nutritional epidemiology

Community health studies and alleged cluster evaluations

Clinical trial support

Chronic diseases, including cancer, cardiovascular disease, and type 2 diabetes

Dietary and lifestyle factors, such as food and supplement intake, smoking behaviors, body weight, and physical activity

Public speaking with a focus on interpreting and articulating epidemiologic evidence

## PROFESSIONAL ACTIVITIES

### Editorial Appointments

2017-Present Guest Editor, *Nutrients*

2015-Present Editorial Board, *American Journal of Clinical Nutrition*

2014–Present Associate Editor, *Frontiers in Nutrition Methodology*

### Peer Reviewer (Abridged List)

*American Journal of Clinical Nutrition*

*American Journal of Epidemiology*

*Epidemiology*

*Journal of the National Cancer Institute*

*Nutrition and Cancer*

*Public Health Nutrition*

*Journal of Food Composition and Analysis*

*Risk Assessment*

*Cancer*

*Cancer Epidemiology Biomarkers and Prevention*

*American Journal of Preventive Medicine*

*European Journal of Cancer Prevention*

*Obesity*

*Southern Medical Journal*

*International Journal of Cancer*

*Journal of Women's Health*

*British Journal of Cancer*

## PUBLICATIONS

### Original Investigations

1. Alexander DD. In Reply I - Prescribing More Stringent Design of Randomized Clinical Trials of Omega-3 Polyunsaturated Fatty Acids. Mayo Clinic Proceedings 2017 Jun;92(6):1006-1007.

2. Alexander DD, Miller PE, van Elswyk M, Kuratko C, Bylsma L. A Meta-Analysis of Randomized Controlled Trials and Prospective Cohort Studies of Eicosapentaenoic and Docosahexaenoic Long Chain Omega-3 Fatty Acids and Coronary Heart Disease Risk. Mayo Clinic Proceedings 2017 Jan;92(1):15-29.

3. Alexander DD, Miller PE, Vargas A, Weed DL, Cohen SS. Meta-analysis of egg consumption and risk of coronary heart disease and stroke. J Am Coll Nutr. 2016 Nov-Dec;35(8): 704-716.

4. Alexander DD, Yan J, Bylsma LC, Northington RS, Grathwohl D, Steenhout P, Erdmann P, Spivey-Krobath E, Haschke F. Growth of infants consuming whey-predominant term infant formulas with a protein content of 1.8 g/100 kcal: a multicenter pooled analysis of individual participant data. Am J Clin Nutr. 2016 Oct;104(4):1083-1092.

5. Maki KC, Guyton JR, Orringer CE, Hamilton-Craig I, Alexander DD, Davidson MH. Triglyceride-lowering therapies reduce cardiovascular disease event risk in subjects with hypertriglyceridemia. J Clin Lipidol. 2016 Jul-Aug;10(4):905-14.

6. Garabrant DH, Alexander DD, Miller PE, Fryzek JP, Boffetta P, Teta MJ, Hessel PA, Craven VA, Kelsh MA, Goodman M. Response to Kay Teschke. Re: Mesothelioma among Motor Vehicle Mechanics: An Updated Review and Meta-analysis. Ann Occup Hyg. 2016 Oct;60(8):1036-7.

7. Alexander DD, Bylsma LC, Elkayam L, Nguyen DL. Nutritional and health benefits of semi-elemental diets: A comprehensive summary of the literature. World J Gastrointest Pharmacol Ther. 2016 May 6;7(2):306-19.

8. J Fryzek, D Alexander, N Summers, J Fraysse, H Reichert, L Townes, J Vanderpuye-Orgle. Indirect Treatment Comparison Of Cabazitaxel For Patients With Metastatic Castration-Resistant Prostate Cancer Who Have Been Previously Treated With A Docetaxel-Containing Regimen. Value in Health. 2016 May 19(3): A139-A140.

9. Alexander DD, Weed DL. On the need for improved methodologic quality of published reviews. Am J Clin Nutr. 2016 Mar;103(3):683-4.

10. Alexander DD, Bylsma LC, Vargas AJ, Cohen SS, Doucette A, Mohamed M, Irvin SR, Miller PE, Watson H, Fryzek JP. Dairy Consumption and Cardiovascular Disease: A Systematic Review and Meta-Analysis. Br J Nutr. 2016 Feb;115(4):737-50.

11. Garabrant DH, Alexander DD, Miller PE, Fryzek JP, Boffetta P, Teta MJ, Hessel PA, Craven VA, Kelsh MA, Goodman M. Mesothelioma among Motor Vehicle Mechanics: An Updated Review and Meta-analysis. Ann Occup Hyg. 2016 Jan;60(1):8-26.

12. Weaver CM, Alexander DD, Boushey CJ, Dawson-Hughes B, Lappe JM, LeBoff MS, Liu S, Looker AC, Wallace TC, Wang DD. Calcium plus vitamin D supplementation and risk of fractures: an updated meta-analysis from the National Osteoporosis Foundation. Osteoporos Int. 2016 Jan;27(1):367-76.

13. Bylsma LC, Alexander DD. A review and meta-analysis of prospective studies of red and processed meat, meat cooking methods, heme iron, heterocyclic amines and prostate cancer. Nutr J. 2015 Dec 21;14(1):125.

14. Alexander DD, Weed DL, Miller PE, Mohamed MA. 2015. Red Meat and Colorectal Cancer: A Quantitative Update on the State of the Epidemiologic Science, J Am Coll Nutr. 2015 Nov-Dec;34(6):521-43

15. Alexander DD, Bassett JK, Weed DL, Barrett EC, Watson H, Harris W. Meta-Analysis of Long-Chain Omega-3 Polyunsaturated Fatty Acids (LCω-3PUFA) and prostate cancer, Nutr Cancer. 2015;67(4):543-54

16. Yurko-Mauro K, Alexander DD, Van Elswyk ME. 2015. Docosahexaenoic Acid and Adult Memory: A Systematic Review and Meta-Analysis. PLoS One. 2015 Mar 18;10(3).

17. Alexander DD, Jiang X, Bylsma LC, Garabrant DH, Irvin SR, Fryzek JP. Historical cancer incidence and mortality assessment in an Illinois community proximal to a former manufactured gas plant. BMJ Open. 2014 Dec 22;4(12).

18. Veruva SY, Steinbeck MJ, Toth J, Alexander DD, Kurtz SM. 2014. Which Design and Biomaterial Factors Affect Clinical Wear Performance of Total Disc Replacements? A Systematic Review. Clin Orthop Relat Res. 2014 Dec;472(12):3759-69

19. Tsuji JS, Perez V, Garry MR, Alexander DD. 2014. Association of low-level arsenic exposure in drinking water with cardiovascular disease: A systematic review and risk assessment. Toxicology 323:78-94.

20. Tsuji JS, Alexander DD, Perez V, Mink PJ. 2014. Arsenic exposure and bladder cancer: quantitative assessment of studies in human populations to detect risks at low doses. Toxicology 317:17-30.

21. Miller PE, Alexander DD, Perez V. 2014. Effects of whey protein and resistance exercise on body composition: a meta-analysis of randomized controlled trials. J Am Coll Nutr 33:163-175.

22. Schmier JK, Miller PE, Levine JA, Perez V, Maki KC, Rains TM, Devareddy L, Sanders LM, Alexander DD. 2014. Cost savings of reduced constipation rates attributed to increased dietary fiber intakes: a decision-analytic model. BMC Public Health 14:374. doi: 10.1186/1471-2458-14-374.:374-14.

23. Miller PE, Van EM, Alexander DD. 2014. Long-chain omega-3 fatty acids eicosapentaenoic acid and docosahexaenoic acid and blood pressure: a meta-analysis of randomized controlled trials. Am J Hypertens 27:885-896.

24. Miller PE, Alexander DD, Weed DL. 2014. Uncertainty of Results in Nutritional Epidemiology. Nutrition Today 49:147-152.

25. Alexander DD. 2013. No association between meat intake and mortality in Asian countries. Am J Clin Nutr 98:865-866.

26. Huhmann MB, Perez V, Alexander DD, Thomas DR. 2013. A self-completed nutrition screening tool for community-dwelling older adults with high reliability: a comparison study. J Nutr Health Aging 17:339-344.

27. Goswami E, Craven V, Dahlstrom DL, Alexander DD, Mowat F. 2013. Domestic asbestos exposure: a review of epidemiologic and exposure data. Int J Environ Res Public Health 10:5629-5670.

28. Alexander DD, Bailey WH, Perez V, Mitchell ME, Su S. 2013. Air ions and respiratory function outcomes: a comprehensive review. J Negat Results Biomed 12:14. doi: 10.1186/1477-5751-12-14.:14-12.

29. Perez V, Alexander DD, Bailey WH. 2013. Air ions and mood outcomes: a review and meta-analysis. BMC Psychiatry 13:29. doi: 10.1186/1471-244X-13-29.:29-13.

30. Alexander DD, Weed DL, Chang ET, Miller PE, Mohamed MA, Elkayam L. 2013. A systematic review of multivitamin-multimineral use and cardiovascular disease and cancer incidence and total mortality. J Am Coll Nutr 32:339-354.

31. Maki KC, Van Elswyk ME, Alexander DD, Rains TM, Sohn EL, McNeill S. 2012. A meta-analysis of randomized controlled trials that compare the lipid effects of beef versus poultry and/or fish consumption. J Clin Lipidol 6:352-361.

32. Kanas GP, Taylor A, Primrose JN, Langeberg WJ, Kelsh MA, Mowat FS, Alexander DD, Choti MA, Poston G. 2012. Survival after liver resection in metastatic colorectal cancer: review and meta-analysis of prognostic factors. Clin Epidemiol 4:283-301.

33. Holscher HD, Czerkies LA, Cekola P, Litov R, Benbow M, Santema S, Alexander DD, Perez V, Sun S, Saavedra JM, Tappenden KA. 2012. Bifidobacterium lactis Bb12 enhances intestinal antibody response in formula-fed infants: a randomized, double-blind, controlled trial. JPEN J Parenter Enteral Nutr 36:106S-117S.

34. Bryan NS, Alexander DD, Coughlin JR, Milkowski AL, Boffetta P. 2012. Ingested nitrate and nitrite and stomach cancer risk: an updated review. Food Chem Toxicol 50:3646-3665.

35. Alexander DD, Weed DL, Mink PJ, Mitchell ME. 2012. A weight-of-evidence review of colorectal cancer in pesticide applicators: the agricultural health study and other epidemiologic studies. Int Arch Occup Environ Health 85:715-745.

36. Alexander DD, Weed DL, Cushing CA, Lowe KA. 2011. Meta-analysis of prospective studies of red meat consumption and colorectal cancer. Eur J Cancer Prev 20:293-307.

37. Alexander DD, Cushing CA. 2011. Red meat and colorectal cancer: a critical summary of prospective epidemiologic studies. Obes Rev 12:e472-e493.

38. Kelsh MA, Alexander DD, Mink PJ, Mandel JH. 2010. Occupational trichloroethylene exposure and kidney cancer: a meta-analysis. Epidemiology 21:95-102.

39. Moolgavkar SH, Turim J, Alexander DD, Lau EC, Cushing CA. 2010. Potency factors for risk assessment at Libby, Montana. Risk Anal 30:1240-1248.

40. Anderson B, Hardin JM, Alexander DD, Grizzle WE, Meleth S, Manne U. 2010. Comparison of the predictive qualities of three prognostic models of colorectal cancer. Front Biosci (Elite Ed) 2:849-56.:849-856.

41. Alexander DD, Wagner ME. 2010. Benzene exposure and non-Hodgkin lymphoma: a meta-analysis of epidemiologic studies. J Occup Environ Med 52:169-189.

42. Alexander DD, Schmitt DF, Tran NL, Barraj LM, Cushing CA. 2010. Partially hydrolyzed 100% whey protein infant formula and atopic dermatitis risk reduction: a systematic review of the literature. Nutr Rev 68:232-245.

43. Alexander DD, Morimoto LM, Mink PJ, Lowe KA. 2010. Summary and meta-analysis of prospective studies of animal fat intake and breast cancer. Nutr Res Rev 23:169-179.

44. Alexander DD, Miller AJ, Cushing CA, Lowe KA. 2010. Processed meat and colorectal cancer: a quantitative review of prospective epidemiologic studies. Eur J Cancer Prev 19:328-341.

45. Alexander DD, Mink PJ, Cushing CA, Sceurman B. 2010. A review and meta-analysis of prospective studies of red and processed meat intake and prostate cancer. Nutr J 9:50. doi: 10.1186/1475-2891-9-50.:50-59.

46. Alexander DD, Cabana MD. 2010. Partially hydrolyzed 100% whey protein infant formula and reduced risk of atopic dermatitis: a meta-analysis. J Pediatr Gastroenterol Nutr 50:422-430.

47. Erdreich LS, Alexander DD, Wagner ME, Reinemann D. 2009. Meta-analysis of stray voltage on dairy cattle. J Dairy Sci 92:5951-5963.

48. Alexander DD, Cushing CA. 2009. Quantitative assessment of red meat or processed meat consumption and kidney cancer. Cancer Detect Prev 32:340-351.

49. Alexander DD, Cushing CA, Lowe KA, Sceurman B, Roberts MA. 2009. Meta-analysis of animal fat or animal protein intake and colorectal cancer. Am J Clin Nutr 89:1402-1409.

50. Mink PJ, Alexander DD, Barraj LM, Kelsh MA, Tsuji JS. 2008. Low-level arsenic exposure in drinking water and bladder cancer: a review and meta-analysis. Regul Toxicol Pharmacol 52:299-310.

51. Mandel JH, Kelsh M, Mink PJ, Alexander DD. 2008. Trichloroethylene exposure and non-Hodgkin's lymphoma: supportive evidence (letter). Occup Environ Med 65:147-148.

52. Kelsh MA, Alexander DD, Kalmes RM, Buffler PA. 2008. Personal use of hair dyes and risk of bladder cancer: a meta-analysis of epidemiologic data. Cancer Causes Control 19:549-558.

53. Alexander DD. 2007. An environmental cause of orofacial cleft defects or an unexplained cluster? South Med J 100:553-554.

54. Alexander DD, Waterbor J, Hughes T, Funkhouser E, Grizzle W, Manne U. 2007. African-American and Caucasian disparities in colorectal cancer mortality and survival by data source: an epidemiologic review. Cancer Biomark 3:301-313.

55. Alexander DD, Mink PJ, Adami H-O, Cole P, Mandel JS, Oken MM, Trichopoulos D. 2007. Multiple myeloma: A review of the epidemiologic literature. Int J Cancer 120:40-46.

56. Alexander DD, Mink PJ, Adami HO, Chang ET, Cole P, Mandel JS, Trichopoulos D. 2007. The non-Hodgkin lymphomas: a review of the epidemiologic literature. Int J Cancer 120 Suppl 12:1-39.

57. Alexander DD, Kelsh MA, Mink PJ, Mandel JH, Basu R, Weingart M. 2007. A meta-analysis of occupational trichloroethylene exposure and liver cancer. Int Arch Occup Environ Health 81:127-143.

58. Mandel JH, Kelsh MA, Mink PJ, Alexander DD, Kalmes RM, Weingart M, Yost L, Goodman M. 2006. Occupational trichloroethylene exposure and non-Hodgkin's lymphoma: a meta-analysis and review. Occup Environ Med 63:597-607.

59. Alexander DD, Mink PJ, Mandel JH, Kelsh MA. 2006. A meta-analysis of occupational trichloroethylene exposure and multiple myeloma or leukaemia. Occup Med (Lond) 56:485-493.

60. Chatla C, Jhala NC, Katkoori VR, Alexander D, Meleth S, Grizzle WE, Manne U. 2005. Recurrence and survival predictive value of phenotypic expression of Bcl-2 varies with tumor stage of colorectal adenocarcinoma. Cancer Biomark 1:241-250.

61. Saif MW, Alexander D, Wicox CM. 2005. Serum Alkaline Phosphatase Level as a Prognostic Tool in Colorectal Cancer: A Study of 105 patients. J Appl Res 5:88-95.

62. Alexander DD, Jhala N, Chatla C, Steinhauer J, Funkhouser E, Coffey CS, Grizzle WE, Manne U. 2005. High-grade tumor differentiation is an indicator of poor prognosis in African Americans with colonic adenocarcinomas. Cancer 103:2163-2170.

63. Alexander DD, Chatla C, Funkhouser E, Meleth S, Grizzle WE, Manne U. 2004. Postsurgical disparity in survival between African Americans and Caucasians with colonic adenocarcinoma. Cancer 101:66-76.

64. Manne U, Alexander DD, Chatla C. 2004. Author Reply: Postsurgical Disparity in Survival between African Americans and Caucasians with Colonic Adenocarcinoma. Cancer 101:2900.

## Oral Presentations

1. Alexander DD. Epidemiology of egg consumption and risk of coronary heart disease and stroke. Webinar: What does the science say? Eggs and heart health. Egg Farmers of Canada. June 6, 2017

2. Alexander DD. Consumption: diet and lifestyle perspective. Meat and Livestock Australia Scientific Workshop. Sydney, Australia. April 10, 2017.

3. Alexander DD. Are red meat consumers unhealthy? Nutrition in Action Symposium. Sydney, Australia. April 5, 2017.

4. Alexander DD. Red meat and cancer risk: interpreting the evidence. NCBA Discovery Symposium. Denver, CO. July 27, 2016.

5. Alexander DD. Theory: bias and confounding. Strengthening Causal Inference in Behavioral Obesity Research. Summer short course; University of Alabama-Birmingham, July 25, 2016.

6. Alexander DD. Red meat and cancer risk: interpreting the evidence. Danish Nutrition Society; University of Copenhagen. Copenhagen, Denmark. June 21, 2016.

7. Alexander DD. Meta-analysis: recycling garbage or an important tool for evaluating the evidence? Drug and Medical Device Seminar. Chicago, IL. May 19-20, 2016.

8. Alexander DD. Evaluating the relationship of meat and cancer risk. Canadian Nutrition Society, Ottawa, Canada. May 5-7, 2016.

9. Alexander DD. Becoming a nutrition detective. Washington State Academy of Nutrition and Dietetics, Annual Conference. Vancouver, WA. April 18, 2016.

10. Alexander DD. Red meat and chronic disease: A closer look into the data. Utah Academy of Nutrition and Dietetics, Annual Conference. Ogden, UT. March 24, 2016.

11. Alexander DD. Meat and cancer risk: understanding the science. Protein: Contributions and Controversies. Toronto, Canada. February 29, 2016.

12. Alexander DD. Understanding the role of epidemiology in disease causation. Asbestos Medicine; DRI. Las Vegas, NV, November 5-6, 2015.

13. Alexander DD. Theory: bias and confounding in observational studies. Strengthening Causal Inference in Behavioral Obesity Research. Summer short course; University of Alabama-Birmingham, July 20, 2015.

14. Alexander DD. Red and processed meat consumption and cancer. International Meat Society. Calgary, Canada. July 1-2, 2015.

15. Alexander DD. Red meat consumption and chronic disease. Canadian Nutrition Society. Winnipeg, Canada. May 30, 2015.

16. Alexander DD. Understanding studies of diet and chronic disease. New Mexico Academy of Nutrition and Dietetics. Albuquerque, NM. April 24, 2015.

17. Alexander DD. Overview of FDA Health Claims and the Submission Process. Webinar. January 13, 2015.

18. Alexander DD. Becoming a Nutrition Detective: Critically Reviewing Research and Communicating Science. DBC Communications Camp, Academy of Nutrition and Dietetics. Las Vegas, NV; January 17, 2015.

19. Alexander DD, State of the epidemiologic science on red meat and chronic disease. Health Canada. Ottawa, Canada; October 22, 2014

20. Alexander DD. Observational epidemiologic studies of breakfast intake. Kellogg Scientific Advisory Board Meeting. Battle Creek, MI; October 1, 2014.

21. Alexander DD. Caffeine intake during pregnancy: the pregnancy signal and reproductive outcomes. The Toxicology Forum, 40th Annual Summer Meeting, Aspen, CO, July 7-10, 2014.

22. Alexander DD. Understanding studies of diet and chronic disease. Delaware Dietetic Association, Dover, DE, May 9, 2014.

23. Alexander DD. Red meat and colorectal cancer: a quantitative update on the state of the science. Experimental Biology, San Diego, CA, April 27, 2014.

24. Alexander DD. Nutrition Detective: An Epidemiologist's Investigation into Diet and Chronic Disease. 31st Annual Health & Nutritional Sciences Conference, South Dakota State University, April 10, 2014.

25. Alexander DD. Summarizing, Interpreting, and Communicating Epidemiologic Evidence. GOED Exchange, Salt Lake City, UT, February 6, 2014.

26. Alexander DD. Synthesizing and Summarizing Epidemiology Evidence, Health Economics, and Fiber and Constipation. Food & Fiber Summit: Identifying Practical Solutions to Meet America's Fiber Needs, Washington DC, January 28, 2014.

27. Alexander DD. Interpreting Epidemiologic Evidence, and a Case Study on Red Meat and Colorectal Cancer. Oncology Nutrition Symposium, Hollywood, FL, January 18, 2014.

28. Alexander DD. OMEGA-3 LC-PUFAs: Judging the Epidemiologic Evidence. GOED Fall Member Meeting at the SupplySide West Tradeshow, Las Vegas, NV, November 14, 2013.

29. Alexander DD. Nutritional Epidemiology: Are We Overstating the Evidence? Missouri Academy of Family Physicians, 21stAnnual Fall Conference, Branson, MO, November 9, 2013

30. Alexander DD. Interpreting Epidemiologic Evidence. DRI Asbestos Medicine Seminar, New Orleans, LA, November 8, 2013.

31. Alexander DD. DRI Research Roundtable: Full-Fat Dairy Products in Nutrition and Health (panel discussant). October 10, 2013.

32. Alexander DD. Update on Red Meat and Colorectal Cancer. International Meat Society Annual Meeting. Granada Spain (webinar), September 14, 2013.

33. Alexander DD. Sustainable Nutrition Roundtable (panel discussant). August 2, 2013.

34. Alexander DD. Dairy and body composition: Making sense of meta-analyses. Dairy Research Institute Meeting: Dairy and Weight, Chicago, IL, June 4–5, 2013.

35. Alexander DD. Nitrate and nitrite exposure and stomach cancer: summary of the epidemiologic evidence. Canadian Nutrition Society, Annual Meeting, Quebec City, Canada, May 31, 2013.

36. Alexander DD. Meta-analysis: Judging the evidence, fish oil and cardiovascular disease. AOCS: Omega-3 Fatty Acids and Heart Health, Montreal, Canada, April 28–May 1, 2013.

37. Alexander DD. Epidemiologic evidence: Investigation Into diet and chronic disease. MINK Conference: Nutrition Without Boundaries, Kansas City, KS, April 6, 2013.

38. Alexander DD. A systematic review of multivitamin use and mortality, cardiovascular disease, and cancer. Council for Responsible Nutrition (CRN): Day of Science, Laguna Beach, CA, October 2–3, 2012.

39. Alexander DD. Diet and cancer: Are we asking the right question? Cancer Society of New Zealand, New Zealand Ministry of Health, Network Communications, Wellington, New Zealand, September 11, 2012.

40. Alexander DD. Interpreting meta-analyses for dietetic practice. Professional development session for New Zealand dietitians, University of Otago, Dunedin, New Zealand, September 10, 2012.

41. Huhmann MB, Kaspar KM, Perez V, Alexander DD, Thomas DR. Accuracy of a new self-completed nutrition screening tool for community-dwelling older adults. Oral Presentation at the European Society for Clinical Nutrition and Metabolism, Barcelona, Spain, September 8–11, 2012.

42. Alexander DD. Interpreting meta-analysis for dieticians in practice. International Congress of Dietetics, Dieticians Association of Australia. Sydney, Australia, September 7, 2012.

43. Alexander DD. Red meat and colorectal cancer: Are we asking the right question(s)? Diet and Gut Health Symposium. Nutrition Society of Australia. Sydney, Australia, September 5, 2012.

44. Alexander DD. Diet and gut health round table meeting and presentation. Meat & Livestock Australia, Sydney, Australia, September 4, 2012.

45. Alexander DD. An update on red meat and cancer. Webinar, International Congress of Meat Science and Technology, Montreal, Canada, August 12–17, 2012.

46. Alexander DD. How to improve the research integrity of meta-analyses and systematic reviews. Scientific Approaches to Strengthening Research Integrity in Nutrition and Energetics, Mohonk Mountain House, NY, August 7–8, 2012.

47. Alexander DD. Sustainable agriculture and the integration of plant- and animal-based foods. California Milk Advisory Board, San Francisco, CA, July 25, 2012.

48. Alexander DD. Nitrate and nitrite exposure and stomach cancer: Summary of the epidemiologic evidence. IFT Annual Meeting, Las Vegas, NV, June 25–28, 2012.

49. Perez V, Schmier JK, Alexander DD. Race/ethnic disparities in pediatric discharges from all US community, non-rehabilitation hospitals for respiratory syncytial virus (RSV) among children one year or younger. Oral presentation at the 45th Annual Society for Epidemiologic Research (SER) Meeting, Minneapolis, MN, June 27–30, 2012.

50. Alexander DD. The nutrition detective: An epidemiologist's look at diet and chronic disease conundrums. New York State Dietetic Association 2012 Annual Meeting & Expo, Albany, NY, May 4–5, 2012.

51. Alexander DD. Epidemiology: Methods for weighing the evidence. MDLA Young Lawyers Meeting, Minneapolis, MN, February 9, 2012.

52. Alexander DD. Nutritional epidemiology: Weighing the evidence and a case study on red meat intake and colorectal cancer. MeatEat Nutritional Conference, Oslo, Norway, September 1, 2011.

53. Alexander DD. Prevalence of bone metastasis from breast, lung or prostate cancer: A systematic and quantitative review of the literature. International Conference on Pharmacoepidemiology, Chicago, IL, August 15–17, 2011.

54. Alexander DD. Benzene epidemiology: Weighing the evidence and a case study of non-Hodgkin lymphoma. Benzene Litigation Conference Audiocast, Chicago, IL, July 13, 2011.

55. Alexander DD. Red meat consumption and colorectal cancer: A meta-analysis of prospective epidemiologic studies. Congress of Epidemiology, Montreal, Canada, June 21, 2011.

56. Alexander DD. Translating the science: Red meat & cancer. Ranch Event 2011, Texas Beef Council, San Antonio, TX, June 2, 2011.

57. Alexander DD. Epidemiology consulting and a case study on red meat and cancer. Distinguished Alumni Investigator Lecture, Birmingham, AL, March 23, 2011.

58. Alexander DD. Nutritional epidemiology: Weighing the evidence. International Life Sciences Institute-ILSI North America Annual Meeting, Orlando, FL, January 24–25, 2011.

59. Alexander DD. The nutrition detective: Translating nutrition science into practice. Texas Dietetics Association. December 8, 2010 (webinar).

60. Alexander DD. The epidemiology of red and processed meat consumption and cancer and cardiovascular disease. The role of red meats in a healthy diet: U.S. Meat Export Federation, Mexico City, Mexico, October 20, 2010 (Keynote speaker).

61. Alexander DD. Meat consumption and cancer: An epidemiologic overview. Live Well, Napa Valley, June 10, 2010.

62. Alexander DD. Red meat consumption and colorectal cancer: A meta-analysis of prospective studies. Experimental Biology, Anaheim, CA, April 26, 2010.

63. Alexander DD. A weight-of-evidence review of colorectal cancer in pesticide applicators: The Agricultural Health Study and other Epidemiologic Studies. CropLife America/Rise Spring Conference, Washington DC, April 15, 2010.

64. Alexander DD. Meat and Cancer. American Meat Institute, Spring Meeting, April 14, 2010.

65. Alexander DD, Weed DL. Ongoing assessment of pesticides and colorectal cancer: A weight of evidence evaluation of epidemiologic literature. Environmental Protection Agency SAP draft framework, Washington DC, February 2, 2010.

66. Alexander DD. Benzene exposure and non-Hodgkin lymphoma: a meta-analysis. Society for Epidemiologic Research, Anaheim, CA, June 24, 2009 (Spotlight Session).

67. Alexander DD. The epidemiology of red and processed meat and cancer. IMS Human Nutrition and Health Committee meeting, Chicago, IL, May 20, 2009 (Invited Speaker).

68. Erdreich LS, Wagner M, Van Kerkhove M, Alexander DD. Stray voltage meta-analysis: needs, methods and challenges. 46th Annual Rural Energy Conference, La Crosse, WI, February 28, 2008.

69. Alexander DD. Epidemiologic evaluation of red meat and cancer. Cattle Industry Convention & Trade Show, Nutrition Roundtable, Reno, NV, February 7, 2008.

70. Alexander DD. Red meat scientific assessment. Industry Stakeholder Cancer Forum, Chicago, IL, October 11, 2007.

71. Alexander DD. Meta-analysis of occupational trichloroethylene exposure and lymphohematopoietic malignancies and liver cancer. Epidemiology Seminar Series, University of Illinois, Chicago, IL, November 17, 2006.

72. Kelsh MA, Mandel JH, Mink PJ, Weingart M, Alexander DD, Goodman M. A meta-analysis of kidney cancer, non-Hodgkin's lymphoma and occupational trichloroethylene exposure. Proceedings, 2nd North American Congress of Epidemiology, Seattle, WA, June 2006.

73. Mink PJ, Alexander DD, Barraj L, Kelsh Ma, Tsuji J. A review and meta-analysis of low-level arsenic exposure in drinking water and bladder cancer. Presentation to the Canadian Pest Management Regulatory Agency, Ottawa, Canada, June 2006.

74. Mandel JH, Alexander DD, Kelsh MA. Occupational trichloroethylene exposure: recent insights from epidemiologic and toxicologic perspectives. State of the Art Conference of the American College of Occupational and Environmental Medicine, Chicago, IL, October 2005.

## Book Chapter

1. Kelsh MA, Alexander DD. Occupational and environmental epidemiology. In: Encyclopedia of Epidemiology. Sage Publications, Thousand Oaks, CA, 2007.

## Abstracts

1. Bylsma L, Alexander DD. A Review and Meta-Analysis of Prospective Studies of Red and Processed Meat, Meat Cooking Methods, Heme Iron, Heterocyclic Amines and Prostate Cancer. Experimental Biology, San Diego, CA, April 2-6, 2016.

2. Miller PE, Alexander DD. A Review and Meta-Analysis of Prospective Studies of Red and Processed Meat and Pancreatic Cancer. Experimental Biology, San Diego, CA, April 2-6, 2016.

3. Althuis M, Alexander DD, Frankenfeld F, Weed DL. Meta-analysis of observational studies in context: sugar-sweetened beverages and type 2 diabetes. Federation of American Societies for Experimental Biology (FASEB). March, 2015

4. Alexander DD, Weed DL. Red meat and colorectal cancer: a quantitative update on the state of the science. Experimental Biology, San Diego, CA, April 26-30, 2014.

5. Alexander DD, Mitchell M, Taylor A, Lowe K, Langeberg W, et al. Prevalence of bone metastasis in breast cancer patients and subsequent survival: A systematic and quantitative review of the literature. San Antonio Breast Cancer Symposium, San Antonio, TX, December 6–10, 2011.

6. Mitchell M, Taylor A, Lowe K, Langeberg W, Alexander DD, et al. Prevalence of bone metastasis from breast, lung or prostate cancer: A systematic and quantitative review of the literature. International Conference on Pharmacoepidemiology, Chicago, IL, August 15–17, 2011.

7. Taylor A, Kanas G, Primrose J, Langeberg W, Alexander DD, et al. Survival after surgical resection of hepatic metastases from colorectal cancer: An updated review and meta-analysis. World Congress on Gastrointestinal Cancer, Barcelona, Spain, June 22–25, 2011.

8. Alexander DD, Perez V, Cushing C, Weed DL. Red meat consumption and colorectal cancer: A meta-analysis of prospective epidemiologic studies. Congress of Epidemiology, Montreal, Canada, June 21, 2011.

9. Perez V, Alexander DD, Cushing C. Processed meat consumption and stomach cancer: A meta-analysis of prospective epidemiologic studies. Congress of Epidemiology, Montreal, Canada, June 21, 2011.

10. Maki KC, Van Elswyk ME, Alexander DD, Rains TM, Sohn EL, McNeill S. A meta-analysis of randomized controlled trials comparing lipid effects of beef with poultry and/or fish consumption. National Lipid Association Annual Scientific Sessions, May, 2011; Abstract 393.

11. Alexander DD. Meta-analysis of prospective epidemiologic studies of red meat intake and colorectal cancer. American Association for Cancer Research, Orlando, FL, April 2–6, 2011.

12. Alexander DD, Cabana MD. Partially hydrolyzed 100% whey protein infant formula and reduced risk of atopic dermatitis: A meta-analysis. Pediatric Academic Societies, Vancouver, BC, Canada, May 1–4, 2010.

13. Alexander DD, Cushing CA. A meta-analysis of red or processed meat intake and prostate cancer. Society for Epidemiologic Research, Anaheim, CA, 2009.

14. Alexander DD, Wagner ME, Kelsh MA. Benzene exposure and non-Hodgkin lymphoma: A meta-analysis. Society for Epidemiologic Research, Anaheim, CA, June 24, 2009.

15. Alexander DD, Schmitt D, Tran N, Barraj L, Cushing CA. Partially hydrolyzed 100% whey infant formula and atopic dermatitis risk reduction: A systematic review of the literature. Experimental Biology, New Orleans, LA, 2009.

16. Alexander DD, Cushing CA, Lowe KL. Meta-analysis of animal fat intake and colorectal cancer. Experimental Biology, New Orleans, LA, 2009.

17. Alexander DD, Cushing CA, Roberts MA. Quantitative assessment of red and processed meat intake and kidney cancer. Experimental Biology, New Orleans, LA, 2009.

18. Lowe KL, Alexander DD, Morimoto LM. Meta-analysis of animal fat intake and breast cancer. Experimental Biology, New Orleans, LA, 2009.

19. Morimoto LM, Alexander DD, Cushing CA. Meta-analysis of red and processed meat consumption and breast cancer. Experimental Biology, New Orleans, LA, 2009.

20. Manne U, Grizzle WE, Alexander DD, Katkoori V. Racial differences in colorectal cancer: the need to educate clinicians and researchers for improved patient care. American Association for Cancer Education, 41st Annual Meeting, Birmingham, AL, October 2007.

21. Gatto NM, Alexander DD, Kelsh MA. A meta-analysis of occupational exposure to hexavalent chromium and stomach cancer. Epidemiology, Sept 2007; Vol 18, issue 5, pS33.

22. Alexander DD, Mink PJ, Mandel JH, Kelsh MA. A meta-analysis of occupational trichloroethylene exposure and multiple myeloma or leukemia. Proceedings 2nd North American Congress of Epidemiology, Seattle, WA, June 2006.

23. Kelsh MA, Mandel JH, Mink PJ, Weingart M, Alexander DD, Goodman M. A meta-analysis of kidney cancer, non-Hodgkin's lymphoma and occupational trichloroethylene exposure. Proceedings, 2nd North American Congress of Epidemiology, Seattle, WA, June 2006.

24. Mink PJ, Alexander DD, Barraj L, Kelsh MA, Tsuji J. Meta-analysis of low level arsenic exposure and bladder cancer. Proceedings, 2nd North American Congress of Epidemiology, Seattle, WA, June 2006.

25. Alexander DD, Mink PJ, Butchko H. How "fast food" is used and interpreted in scientific research: methodological considerations. Proceedings, Experimental Biology 2006, San Francisco, CA, April 2006.

26. Kapica CM, Alexander DD, Mink PJ, Butchko H. The definition of fast food in published studies. Proceedings, Experimental Biology 2006, San Francisco, CA, April 2006.

27. Alexander D, Chatla C, Funkhouser E, Jhala N, Grizzle WE, Manne U. Racial differences in survival based on tumor differentiation and stage in patients who have undergone surgery for colon cancer. J Clin Oncol 2004; 22:14S (July Supplement).

28. Alexander D, Funkhouser E, Saif M. Alkaline phosphatase (AP) as a prognostic tool in colorectal cancer (CRC). Proceedings, American Society of Clinical Oncology 2003; 22:354.

29. Malhotra P, Kallergi M, Alexander D, et al. Discrepancies between film and digital mammography interpretations. Medical Imaging 2002, Proceedings of SPIE (The International Society for Optical Engineering), February 2002.

30. Alexander D, Malhotra P, Kallergi M, et al. Digital vs. film mammography: calcification interpretation. American Association of Physicists in Medicine 2001, (July Supplement).

**Poster Presentations**

1. Fryzek J, Alexander DD, Summers N, Fraysse J, Reichert H, Townes L, Vanderpuye-Orgle J. Indirect treatment comparison of cabazitaxel for patients with metastatic castration-resistant prostate cancer who have been previously treated with a docetaxel-containing regimen. International Society for Pharmacoeconomics and Outcomes Research (ISPOR). Washington DC. May 21-25, 2016

2. Miller PE, Alexander DD. A Review and Meta-analysis of Prospective Studies of Red and Processed Meat and Pancreatic Cancer. Experimental Biology. San Diego, CA. April 4, 2016.

3. Maki KC, Guyton JR, Orringer CE, Hamilton-Craig I, Alexander DD, Davidson MH: Triglyceride-lowering therapies reduce cardiovascular disease event risk in subjects with hypertriglyceridemia. National Lipid Association Scientific Sessions, Chicago, IL, USA, June 11–14, 2015

4. Bylsma L, Miller P, Alexander DD. Meta-analysis of red meat intake and type 2 diabetes. Experimental Biology. Boston, MA, March 31, 2015.

5. Tsuji JS, Alexander DD, Perez V. Low-level arsenic in drinking water and bladder cancer risk: Meta-analysis update and risk assessment implications. Annual Meeting of the Society of Toxicology, San Antonio, TX, March 10–14, 2013.

6. Perez V, Alexander DD, Bailey WH. Air ions and mood outcomes: A review and meta-analysis. Poster presentation at the American College of Epidemiology, Chicago, IL, September 8–11, 2012.

7. Huhmann MB, Kaspar KM, Perez V, Alexander DD, Thomas DR. Accuracy of a self-completed nutrition screening tool for community-dwelling older adults when completed by the patient or caregivers. Poster presentation at the International Academy on Nutrition and Aging Meeting, Albuquerque, NM, July 12–13, 2012.

8. Perez V, Schmier JK, Alexander DD. Race/ethnic disparities in pediatric discharges from all US community, non-rehabilitation hospitals for respiratory syncytial virus (RSV) among children one year or younger. Oral presentation at the 45th Annual Society for Epidemiologic Research (SER) Meeting, Minneapolis, MN, June 27–30, 2012.

9. Alexander DD, Mitchell M, Taylor A, Lowe K, Langeberg W, et al. Prevalence of bone metastasis in breast cancer patients and subsequent survival: A systematic and quantitative

review of the literature. San Antonio Breast Cancer Symposium, San Antonio, TX, December 6–10, 2011.

10. Alexander DD, Perez V, Cushing C, Weed DL. Red meat consumption and colorectal cancer: A meta-analysis of prospective epidemiologic studies. Congress of Epidemiology, Montreal, Canada, June 21, 2011.

11. Perez V, Alexander DD, Cushing C. Processed meat consumption and stomach cancer: A meta-analysis of prospective epidemiologic studies. Congress of Epidemiology, Montreal, Canada, June 21, 2011.

12. Gatto NM, Alexander DD, Kelsh MA. A meta-analysis of occupational exposure to hexavalent chromium and stomach cancer. 19th Annual International Society of Environmental Epidemiology Conference, Mexico City, Mexico, September 5–9, 2007.

13. Alexander DD, Mink PJ, Mandel JH, Kelsh MA. A meta-analysis of occupational trichloroethylene exposure and multiple myeloma or leukemia. Proceedings, 2nd North American Congress of Epidemiology, Seattle, WA, June 2006.

14. Mink PJ, Alexander DD, Barraj L, Kelsh MA, Tsuji J. Meta-analysis of low level arsenic exposure and bladder cancer. Proceedings, 2nd North American Congress of Epidemiology, Seattle, WA, June 2006.

15. Alexander DD, Mink PJ, Butchko H. How "fast food" is used and interpreted in scientific research: methodological considerations. Experimental Biology 2006, San Francisco, CA, April 2006.

16. Kapica CM, Alexander DD, Mink PJ, Butchko H. The definition of fast food in published studies. Experimental Biology 2006, San Francisco, CA, April 2006.

17. Mink PJ, Alexander DD, Barraj LM, Kelsh MA, Tsuji JS. Meta-analysis of low level arsenic exposure and bladder cancer: implications for risk assessment in the United States. 45th Annual Meeting of the Society of Toxicology, San Diego, CA, March, 2006.

18. Alexander D, Jhala N, Chatla C, Steinhauer J, Funkhouser E, Coffey C, Grizzle WE, Manne U. Racial differences in survival based on tumor differentiation and stage in patients who have undergone surgery for colon cancer. The 2004 American Society of Clinical Oncology Annual Meeting, New Orleans, LA, June 2004.

19. Chatla C, Alexander D, Manne U. Prognostic significance of Bcl-2 expression and p53 nuclear accumulation based on nodal status in patients with colorectal adenocarcinoma. The 95th Annual meeting of the American Association for Cancer Research, Orlando, Florida, March 2004.

20. Alexander D. Post-surgical disparity in survival between African-Americans and Caucasians with colonic adenocarcinomas. The UAB Comprehensive Cancer Center Annual Research Retreat, Birmingham, Alabama, October 2003.

21. Malhotra P, Kallergi M, Alexander D, et al. Discrepancies between film and digital mammography interpretations. The annual meeting for Medical Imaging: Observer Performance Studies, San Diego, CA, February 26–28, 2002. (Poster Presentation, Presenter: P. Malhotra).

22. Alexander D, Malhotra P, Kallergi M, et al. Digital vs. film mammography. The American Association of Physicists in Medicine Conference, Salt Lake City, UT, July 22–26, 2001.